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BLOOD CULTURE IN TUBERCULOSIS.

By W. J. PENFOLD, M.B., Ch.M. (Edinburgh), D.P.H., B.Hyg. (Durham), M.R.C.S. (England), L.R.C.P. (London),

AND

HILDRED M. BUTLER, B.Sc. (Melbourne),

(From the Baker Institute of Medical Research, Alfred Hospital, Melbourne.)

THE recognition of the presence in the blood of the infecting agent of tuberculosis was first effected by Villemin (1868), who transferred blood from tuberculous rabbits and patients to normal rabbits and so infected them with tuberculosis. This work was confirmed by several authors, but apparently was not used for diagnostic purposes in clinical practice. The next important step was taken by Weichselbaum (1884), who, by microscopic examination of the blood of three patients dying of miliary tuberculosis, detected the presence of acid-fast organisms. Innumerable modifications of this direct smear method have been made, which are reviewed by Calmette[1] in his monograph (1923). The most successful worker with the dried smear method may have been Rosenberg, who applied it in 312 cases of tuberculosis and found each case positive. Löwenstein (1932),[2] however, in a comparison of the direct smear method with the cultural method in a series of 176 cases found that in eight cases both methods yielded positive results; in 45 cases the culture only gave a positive result, while in one case the smear only showed the presence of acid-fast bacilli. Villemin's work then, and that of others, had shown that tuberculosis was sometimes associated with a bacillæmia, and in 1917 Mildred Clough[3] was able to cultivate the organisms from the blood of each of five cases of miliary tuberculosis. Bingold (1928)[4] reported four positive cultures in a series of 370 cases of pulmonary tuberculosis.

The more general question of the cultivation of tubercle bacilli from the blood in all forms of tuberculosis has been most intensively worked at by E. Löwenstein, of Vienna. He worked at the problem from 1905, but did not develop a method satisfactory to himself before 1927. He (1931)[5] criticized unfavourably the usual methods of cultivating tubercle bacilli from the blood. He recommended asparagin egg medium for the purpose, the treating of the blood with 3% acetic acid, and the use for cultivation of the washed deposit freed from hæmoglobin. He claimed that in these circumstances cases of miliary tuberculosis, tuberculous meningitis, and advanced pulmonary tuberculosis invariably gave positive blood cultures. Of eight cases of afebrile extrapulmonary tuberculosis he found the blood cultures positive in six.

In his subsequent papers Löwenstein deals with the frequency of bacillæmia in the various forms of tuberculosis. An outline of the method is given repeatedly by him and, apart from minor points, the only variation of note is the recommendation of distilled water instead of acetic acid to effect the lysis of the red cells.[6][7] He stated that in moderately severe pulmonary tuberculosis the blood cultures were positive in 15% of the cases, while in slight pulmonary infections the blood cultures were invariably negative.[8] By September, 1930, Löwenstein[9] had reported 54 positive blood cultures obtained from 140 patients suffering from pulmonary tuberculosis.

In 1930 ten cases of cutaneous tuberculosis were reported by him as giving uniformly positive blood culture results.[8] Subsequent reports on this same subject by Löwenstein and his colleagues show many fewer successes. In 1931 Kren and Löwenstein[10] gave an account of blood cultures in 51 cases of skin tuberculosis, 38 of which gave positive results for tubercle bacilli. In 1932, Kissmeyer and Löwenstein[6] gave the results of 255 blood cultures from patients with skin tuberculosis. Forty-nine of these cultures gave a positive result. Blood culture was attempted in 98 cases of non-tuberculous skin disease, 10 of which gave a positive finding. In 2 of the 10 cases lung tuberculosis was found, but in the other 8 tuberculosis had not been demonstrated at the time of publication of the article. This seems curious, since Löwenstein had previously done 3,000 control blood cultures without finding any positive results.

Lichtenstern (1931),[11] working with Löwenstein, concluded that the blood culture method was of definite help in the diagnosis of uro-genital tuberculosis. He regarded it as superior to the examination of the urine for tubercle bacilli. In each of 10 cases of fistula after operation he found that blood culture gave positive results. All blood cultures gave negative results in 27 cases of uro-genital disease not suspected of being tuberculous.

In 1931, Löwenstein and Wessely[12] claimed that a tuberculous bacillæmia was of frequent occurrence in laryngeal tuberculosis. Of 47 cases, 55% yielded a growth of tubercle bacilli from the blood, and blood culture was only carried out once in each of these cases. Löwenstein insisted on the need for repeated attempts at blood culture and believed that in this way a higher percentage of positive results would be obtained. He stated that in intestinal tuberculosis the bacillæmia was demonstrable in 90% of cases.[8] He found also that the number of positive cultures was high in tuberculosis of the eye.[9]

Löwenstein had not neglected control blood cultures. He claimed in 1931 that in more than 3,000 healthy individuals he had regularly failed to cultivate tubercle bacilli from the blood. He likewise failed in cases of gonorrhœa, syphilis, carcinoma, psoriasis, septic processes, pneumonia, typhus *et cetera*.[13] He discussed the value of his results from the points of view of diagnosis and prognosis and compared them with those obtained by other methods. He found, for example, in uro-genital cases that, as the healing commenced, the bacilli disappeared from the blood. The same applied in cases of tuberculosis of bone. He regarded his blood culture method as preferable in diagnosis to the tuberculin reaction. He believed a definite bacillæmia to exist as long as a tuberculous lesion was active.

Löwenstein and Reitter have recorded remarkable results obtained from blood cultures in rheumatic fever. In their early publications they reported a series of twenty-one cases in which each case yielded a positive blood culture for tubercle bacilli. Their later work showed that there was some evidence that positive blood cultures in rheumatic fever were associated with fever and increased activity on the part of the disease. One case is reported in which they were able to cultivate tubercle bacilli from the umbilical cord of an infant born of a mother suffering at the time of delivery from acute rheumatism. These various points are dealt with in papers.[9][14][15][16] Löwenstein, in collaboration with Amersbach and A. Löwenstein,[17] showed that in 14 rheumatics examined during afebrile periods no tubercle bacilli could be cultivated from the blood, though they cultivated them from the tonsils of 5 of the patients. Histologically the tonsils showed no specific signs of tuberculous infection.

In each of 4 cases of chorea Löwenstein claimed to have obtained a positive blood culture of tubercle bacilli. Similarly he claimed to have cultivated tubercle bacilli from the blood of 17 out of 40 patients with multiple sclerosis. He admitted, however, that in 7 of these 17 cases the bacillæmia might have been due to lesions in the lungs. Tubercle bacilli were grown by him from the blood in 9 out of 24 cases of *dementia præcox*, and 4 out of 6 cases of retrobulbar neuritis. In one of the last mentioned cases avian tubercle bacilli were recovered both from the blood and the tonsils.[11][15]

In 1932, Löwenstein[7] recorded the blood culture findings in nine cases of asthma. All gave him blood cultures positive in respect of tubercle bacilli.

In one of his most recent articles Löwenstein[18] has compared the value of his method of blood culture in tuberculosis with the guinea-pig inoculation test. He claimed that his cultural test was much superior to the animal test, but this paper is disfigured by so many inaccuracies that it is quite impossible to make a valid comparison from the data given. For instance, in Table III of his paper cultural results are given which apparently were obtained before the specimens reached the laboratory (see cases, Reitermeyer, Exner and Kranner). The virulence test of strain Lindenthal was carried out on July 9, 1930, though the animal (hen) is stated to have died on May 18, 1930. In Table VI positive cultural results were obtained in March, 1932, though the paper was published in February, 1932. Similar errors occur throughout. In other of Löwenstein's papers there are discrepancies between the figures given in the text and those of the tables. The large amount of work he has carried out on this subject merited more care in the presentation of the results.

Other of Löwenstein's collaborators, Fischer and Urgoiti,[19][20] Jerusalem and Urgoiti,[21] and Konrad,[22] have reported high percentages of positive results similar to those of Löwenstein himself.

Apart from Löwenstein and his collaborators, the German literature gives an account of the work of many other authors, none of whom has been able to reproduce Lowenstein's large number of positive results in the living subject.

Popper, Bodart and Schindler (1931)[23] reported a high percentage of positive results from blood obtained *post mortem* from fatal cases of tuberculosis. But with blood obtained during life they had very little success. In a later communication (1932)[24] they gave the results of more than 600 blood cultures taken either during life or *post mortem*. Forty-six out of 191 cultures made *post mortem* from the heart blood of tuberculous and non-tuberculous cases yielded positive results. The positive cultures were all from cases of miliary or pulmonary tuberculosis, except one which was from a case of genito-urinary tuberculosis.

Blood cultures taken *post mortem* were also carried out by these authors in 22 cases of endocarditis. Of these, 10 were proved *post mortem* to be rheumatic in character. From only one of the bloods of these 10 cases was a strain of tubercle bacillus grown. It formed macroscopically visible colonies (a macroscopic positive). From the surface of the slopes of three other cultures acid-fast bacilli could be identified in smears, but these failed to produce definite growth (microscopic positives). Löwenstein included both these types amongst his positives.

Four hundred and thirty-five blood cultures were made from patients during life. Three hundred and sixty-two of these patients were suffering from tuberculosis or from diseases which Löwenstein regarded as probably tuberculous. Though 46 microscopically positive results were recorded, no macroscopically positive ones were obtained. Five of these microscopic positives occurred in the control non-tuberculous group. None of these microscopically positive cultures yielded growth on subculture.

In the whole series 12 strains of non-pathogenic acid-fast bacilli were isolated. These organisms had the general characteristics of saprophytic acid-fast bacilli.

Axen (1932)[25] failed to obtain positive cultures from 20 cases of tuberculosis. He does not believe that either a permanent or periodic tuberculous bacillæmia occurs in man.

Busson (1931)[26] compared Löwenstein's method with that of animal inoculation and concluded that the latter method, using washed sediment instead of whole blood, was the more reliable.

Jontofsohn[27] obtained results similar to those of Saenz, which are dealt with later, while Unverricht and Dosquet,[28] Bingold and Spier,[29] Kallos,[30] Dettling,[31] all had no or very few positive results with Löwenstein's technique.

The French literature contains reports of several workers who have attempted to confirm Löwenstein's findings.

Saenz (1931)[32] recorded only 18 positive findings in 300 cases of tuberculosis and rheumatism. He did not attach much value to Löwenstein's medium; in fact he abandoned it. Of the strains isolated by him, one, from a case of lupus, was of the avian type, and one presented the characteristics of *B. paratuberculosis*. The remaining strains were of the human type. In a more recent paper (1932)[33] he reported 200 further cases. Of these only 4% yielded positive blood cultures. Domingo (1931)[34] published results similar to those of Saenz. In 172 cases of tuberculosis he got 8 positive blood cultures. In 8 cases of subacute articular rheumatism he found tuberculous bacillæmia twice. In 35 controls, healthy or with other diseases, the cultures were negative.

Nanu, Jonnesco, and Sefanesco (1931)[35] showed the blood to be infected with tubercle bacilli in 2 cases out of 21. These 21 cases were either tuberculous or cases of acute rheumatic fever. One positive was obtained from each group.

English workers have also investigated this problem. Weatherall (1932)[36] in 30 pulmonary cases, and Cumings (1932)[37] in 15 cases of tuberculosis, in some cases of disseminated sclerosis and in lymphadenoma, obtained only negative results.

Schwabacher (1931)[38] reported one success in 11 attempted blood cultures. The positive result was obtained in a pulmonary case; the nature of the negative cases is not discussed. In 1933[39] she reported the results of 403 blood cultures from living patients, 281 of which were tuberculous. These cultures were carried out after a period of study in Löwenstein's laboratory, and every attempt was made to follow his technique exactly.

She obtained three macroscopically visible cultures of tubercle bacilli of human type, and two only microscopically visible cultures. In these latter no macroscopic growth occurred even on subculture. Seven macroscopically visible cultures of

chromogenic acid-fast bacilli were also encountered. All other cultures were negative. The three macroscopically positive cultures were from cases of advanced pulmonary tuberculosis.

One microscopically positive culture occurred among the cultures from the 122 non-tuberculous patients. This series included in its various groups 39 cases of rheumatism, 31 of chorea and 23 of disseminated sclerosis.

Attempts on the part of American workers to confirm Löwenstein's results have met with no greater success than those of European workers. Shapiro,[40] in 167 cases of tuberculosis, obtained positive results in 4·2%. In a study of 28 cases of rheumatic fever and chorea he was unable to confirm Löwenstein's and Reitter's results. He found the technique highly satisfactory with tuberculous guinea-pigs. He concluded that tuberculous bacillæmia was rare, but thought the method might be of diagnostic value in very young children. Cohn (1933)[41] obtained two strains of non-pathogenic acid-fast bacilli in 100 blood cultures from patients with advanced tuberculosis.

Norton and Broom (1933)[42] failed to isolate tubercle bacilli from the blood of 175 patients suffering from tuberculosis. Their technique was not identical with that of Löwenstein.

Troisier and Monaldi (1932),[43] in 76 febrile non-tuberculous cases, obtained from the blood three strains of tubercle bacilli of the human type. One patient suffered from articular rheumatism with streptococcal septicæmia, one from pneumonia and the other from meningococcal meningitis. These authors are of the opinion that tubercle bacilli may be mobilized in the blood during the course of an acute non-tuberculous disease and are not of necessity pathogenic for the host.

Mach and Mach (1932)[44] reported three non-tuberculous cases in which the bacilli had been demonstrated in the blood stream by culture. In two of these cases the organism was stated to have been obtained by Löwenstein himself.

Konrad[22] also records a positive result from a patient believed to be free from tuberculosis.

This section of the work must be extremely difficult, in view of the widespread presence of latent foci in the human subject. These might well be lighted up by other diseases and so vitiate the control results.

The most striking feature of the work on tuberculous bacillæmia is the discrepancy between Löwenstein's results and those obtained by other workers. No independent worker has succeeded in completely confirming Löwenstein's work. This aspect of the work is dealt with in considerable detail by Wilson (1933).[45] As he points out, the determination of the probable frequency of tuberculous bacillæmia is difficult, largely owing to the failure of many workers in this field to differentiate between macroscopically positive results and those microscopically positive; in the latter type macroscopic colonies failed to develop either on primary cultures or on subcultures. We consider those cultures only truly positive in which definite macroscopically visible colonies develop either directly or on subculture. If, on scraping the surface of the original medium after its prolonged incubation and making a smear of the material so obtained, acid-fast bacilli are formed but do not grow on subculture, then we have considered these as negative results. We have assumed that such bacilli were probably present in the original sample of blood taken, but we do not consider them to be *Bacillus tuberculosis*. We are compelled to leave their exact identity to future investigation.

In a great deal of the work it is impossible to tell whether strains isolated were true tubercle bacilli or not. Popper, Bodart and Schindler, Saenz, Rabinowitsch-Kempner, Schwabacher, Legezynski and Ostrowski, Tiedemann, Marchesani, Holm and Norton and Broom all record the finding of saprophytic or atypical tubercle bacilli in blood cultures, so that it seems probable that some at least of the high percentage of positive results recorded by Löwenstein may be due to the inclusion not only of microscopic positive results, but also to the inclusion of saprophytic acid-fast strains.

Owing to Löwenstein's failure to take precautions to guard against these errors, we agree with Wilson that Löwenstein's conclusions cannot be accepted by scientific workers. Löwenstein reported positive results in from 39% to 100% in various types of tuberculosis. The corresponding average figures obtained by other workers in this field vary from nil to 20·6%.

Löwenstein claims to have demonstrated a tuberculous bacillæmia in 67% of patients with acute articular rheumatism and polyarthritis. Subsequent workers who have attempted to confirm this finding average only 6·7% of positive blood cultures. This figure includes both microscopically and macroscopically positive results and is therefore probably too high.

The Authors' Investigations.

The work to be recorded in the present paper was undertaken to examine the practical character of Löwenstein's technique in the recognition of tuberculous bacillæmia. The essence of his technique consists in lysing the blood and centrifuging off the stromata. The latter material has associated with it the tubercle bacilli. This deposit is washed and spread over the surface of Löwenstein's medium. Löwenstein has used distilled water, acetic acid, and saponin for the lysing of the red cells, but in his most recent papers he advocates the use of distilled water.[4][5]

We have used acetic acid and distilled water, but have found that with acetic acid it was more difficult to get rid of the hæmoglobin entirely than was the case with distilled water.

In each of the cases the sediment was divided into two fractions, one portion being treated with sulphuric acid, while the other was not. In the positive cases the positive result was obtained with both fractions. Our results are summarized in Table I.

TABLE I.

TABLE I.

Serial Number.	Diagnosis.	Basis of Diagnosis.	Stage and Intensity of Disease.	Result of Blood Culture.	Interval between Taking Culture and Detecting Acid-Fast Bacilli.	Method of Detecting Growth.	Nature of the Growth.	Virulence.	
								Guinea-pig.	Rabbit.
1	Pulmonary tuberculosis.	(1) Sputum: tubercle bacilli present. (2) *Post mortem* findings.	Advanced with cavity formation and calcification.	(1) Negative. (2) Negative. (3) Positive.	4½ weeks.	Macroscopic.	Good growth on primary and sub-cultures.	Six weeks after subcutaneous injection acid-fast bacilli detected in ulcerated inoculation site and in caseous inguinal glands.	No gross lesions found *post mortem* six months after intravenous inoculation.
2	Pulmonary tuberculosis, encysted empyema right side.	X ray findings.	Cough for one year. Moderate severity.	(1) Negative. (2) Negative.					
3	Pulmonary tuberculosis.	Clinical. (*a*) Hæmoptysis. (*b*) Marked dulness over apex. (*c*) Three members of family died from tuberculosis.	Four years' duration. Mild.	(1) Negative.					
4	Pulmonary tuberculosis.	Sputum: tubercle bacilli present.	Fivemonths' duration, but history of cough with sputum for years. Advanced emaciation, extensive dull areas.	(1) Negative. (2) Negative.					
5	Pulmonary tuberculosis.	Sputum: tubercle bacilli present.	Six months' duration. Moderate severity.	Negative.					
6	Pulmonary tuberculosis.	(1) Sputum: tubercle bacilli present. (2) X ray findings.	Clinically five weeks only. Moderate severity.	(1) Negative. (2) Negative.					
7	Pulmonary tuberculosis.	Sputum: tubercle bacilli present.	Nine years' duration. Moderate severity.	Negative.					
8	Pulmonary tuberculosis.	Sputum: tubercle bacilli present.	Clinically six weeks only. Severe, with cavity formation.	(1) Negative. (2) Negative.					
9	Pulmonary tuberculosis.	Sputum: tubercle bacilli present.	Four months' duration, but history of cough, with sputum, for years.	(1) Negative. (2) Negative.					
10	Broncho-pneumonia.	X ray showed consolidation in lower left lung. Cavity present. Possibly tuberculous.	Clinically two weeks only. Moderate severity.	(1) Positive.	3½ weeks.	Microscopic.	Scanty growth on primary culture, small colonies of acid-fast bacilli on first sub-culture.	Three months after subcutaneous injection acid-fast bacilli detected in ulcerated inoculation site and in caseous inguinal glands. No other gross lesions detected.	No gross lesions found *post mortem* six months after intravenous inoculation.
11	Acute miliary tuberculosis and tuberculous meningitis.	X ray and cerebro-spinal fluid findings.	Four weeks' duration. Acute.	(1) Positive.	6½ weeks.	Macroscopic.	A few colonies on primary cultures, good growth on sub-cultures.	Three months after subcutaneous injection acid-fast bacilli detected at inoculation site, in caseous inguinal and mesenteric glands and in spleen.	No gross lesions found *post mortem* three months after intravenous inoculation.
12	Pulmonary tuberculosis and tuberculous meningitis.	(1) Cerebro-spinal fluid: tubercle bacilli present. (2) *Post mortem* findings.	Three weeks' duration. Acute.	(1) Negative. (2) Negative.					

TABLE I.—*Continued.*

Serial Number.	Diagnosis.	Basis of Diagnosis.	Stage and Intensity of Disease.	Result of Blood Culture.	Interval between Taking Culture and Detecting Acid-Fast Bacilli.	Method of Detecting Growth.	Nature of the Growth.	Virulence.	
								Guinea-pig.	Rabbit.
13	*Lupus vulgaris.*	Clinical.	Nineteen years' duration. Quiescent.	(1) Doubtful Positive.	Five weeks.	Microscopic.	In six months macroscopic colonies did not appear on primary or sub-cultures.		
14	*Lupus vulgaris.*	Clinical.	Two months' duration. Active.	(1) Negative.					
15	Tuberculous ulceration of neck.	Pus: tubercle bacilli present.	Two years' duration. Progressive lesion.	(1) Negative.					
16	Inflamed tubercular appendix.	Biopsic.	Commencement of disease, confined to a thickened appendix.	(1) Negative.					
17	Tuberculosis of spine.	Clinical.	Fifteen years. Quiescent.	Negative.					
18	Tuberculosis of spine; abscess.	(1) Pus: tubercle bacilli present. (2) X ray findings.	Twelve months. Active.	(1) Negative.					
19	Tuberculosis of elbow joint.	Biopsic.	Four and a half years' duration. Active.	Negative.					
20	Tuberculous arthritis of ankle.	Pus: tubercle bacilli present.	Two years' duration. Active.	Negative.					

Blood cultures by the Löwenstein technique have been attempted by us in twenty tuberculous patients. We obtained three positive results. The three strains cultivated have been found to be human in character.

In a fourth case acid-fast rods were seen in smears from the primary cultures and in one subculture, but even after six months' incubation macroscopic colonies failed to develop either on primary culture or on subculture.

Blood cultures were also carried out in three patients with rheumatoid arthritis, two with rheumatic fever, three with chorea, and in six cases of doubtful tuberculosis. None of these cultures yielded acid-fast bacilli.

Table I shows the diagnosis and the clinical condition of each of the twenty cases in which blood culture was attempted; also the nature of the positive findings. The attempt at blood culture was repeated in seven of the cases.

The three strains of acid-fast bacilli isolated conformed in their cultural, morphological and virulence characteristics to the human type. As can be seen from the table, all three strains were of only moderate virulence for the guinea-pig and appeared to be practically non-virulent for the rabbit.

Summary.

In three out of twenty cases of tuberculosis it was possible to cultivate *Bacillus tuberculosis* from the blood by Löwenstein's method. In the cultures from one other case acid-fast organisms could be identified microscopically by means of smears made from the surface of the medium; but these cultures, though incubated for six months, failed to produce colonies visible to the naked eye. Subcultures from them likewise failed to grow.

Conclusion.

The Löwenstein method of blood culture in tuberculosis is troublesome and time-consuming in execution and gives, in our experience, so few positive results that we cannot believe that it is destined to render any great service to the clinician in the diagnosis of tuberculosis.

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ECLAMPSIA.[1]

By ALFRED J. GIBSON, M.B., Ch.M. (Sydney), F.R.A.C.S.,
Honorary Surgeon, The Women's Hospital, Crown Street; Tutor in Obstetrics, The University of Sydney.

IN 1922, at a British Congress of Obstetrics and Gynæcology, a series of reports on cases of eclampsia was presented and discussed. These reports were based on cases observed and treated in different parts of Great Britain. The object was to show upon what lines the disease had actually been treated during the preceding ten years in the different parts of the country and what were the results.

I thought that it might be of interest to collect a series of cases of eclampsia occurring at the Women's Hospital, Crown Street, to analyse them along similar lines, and to compare the results, in order to see whether eclampsia presented any marked variations in Australia as compared with Great Britain and other countries in the northern hemisphere, and to see how the results of treatment compared.

This series comprises all the patients with eclampsia treated at the Women's Hospital from January, 1924, to June 30, 1933. I should like to thank all my colleagues on the staff for the permission to use the records of the patients treated by them.

This period was selected because the records during the past few years contain more detail, and it was necessary to secure at least one hundred cases for comparison. Stroganoff, the great authority on eclampsia, states that in order to form exact conclusions it is necessary to have three hundred observations, or at least two hundred. I admit that this series is a small one, but my time for preparation was limited, and I feel that the conclusions reached should have some value, as they represent a period of nine and a half years, and are being compared with a series of 2,005 British cases.

Of 16,903 obstetric patients admitted, 770 suffered from albuminuria of pregnancy and 101 from eclampsia. This gives an incidence of 1 in 167, or 0·61%, as compared with 1 in 125, or 0·8%, estimated by Eden in Great Britain. It also shows

[1] Read at the annual reunion of the Royal Prince Alfred Hospital Medical Officers' Association, Sydney, September, 1933.

TABLE I.

Showing Incidence of Eclampsia at the Women's Hospital, Crown Street, January, 1924, to June 30, 1933.

Group.	Number.
Obstetric patients admitted	16,903
Albuminuria of pregnancy	770
Eclampsia	101

that the liability of patients with severe albuminuria to develop eclampsia is in the proportion of 1 to 7 or 8.

TABLE II.

Showing number of patients with eclampsia admitted in each year.

Year.	Number of Patients Admitted.	Number of Deaths.
1924	13	2
1925	8	1
1926	10	1
1927	11	2
1928	8	0
1929	9	0
1930	17	1
1931	12	2
1932	8	0
1933 (to June 30)	5	0
Total	101	9

The variation in the numbers admitted each year is a well known fact and is common to all countries. Figures from different countries are shown in Table III.

TABLE III.

Showing incidence of eclampsia in various countries.

Author.	Labours.	Cases of Eclampsia.	
		Number.	Percentage.
Goldberg (Dresden, 1891)	10,717	81	0·75
Cassamayor (Paris, 1892)	16,225	99	0·61
Veit (Germany, 1906)	149,366	905	0·61
Reinberg (Paris, 1905)	26,511	90	0·34
Lichenstein (Leipzig, 1911)	14,836	400	2·68
Williams (Baltimore, 1912)	11,000	110	1·00
McPherson (New York, 1922)	120,000	890	0·75
Hinselmann (Bonn, 1914)	22,716	166	0·73
Cruickshank (Glasgow, 1922)	23,630	814	3·44
Women's Hospital, Crown St. (1933)	16,903	101	0·61

The monthly incidence of the cases at the Women's Hospital is shown in Table IV.

The seasonal incidence is as follows:

Spring (September, October, November), 24 cases	41 cases, 3 deaths. Mortality, 7·3%.
Summer (December, January, February), 17 cases	
Autumn (March, April, May), 26 cases	60 cases, 6 deaths. Mortality 10·0%.
Winter (June, July, August), 34 cases	

The number of deaths for each season is shown in Table V.

TABLE V.

Season.	Number of Deaths.	Mortality Rate.
Spring	2	8·3%
Summer	1	5·8%
Autumn	1	3·8%
Winter	5	14·7%

Table III shows the incidence over a period of years in various countries. Sydney is in the lowest group but one. The table is compiled from hospital statistics and it shows how very prevalent the disease is in Glasgow compared with all the other countries, with the one exception of Leipzig. Baltimore also has a high incidence.

Table IV gives the number of patients with eclampsia admitted to the Women's Hospital during each month of the year. Considerable variation is seen. The greatest number of admissions was in the months of August, March and July. On analysing the results according to the seasons, it is seen that in summer 17 cases occurred, in spring 24, in autumn 26, in winter 34. If we compare the warmer months of summer and spring with the colder months of autumn and winter, we find that eclampsia appears to be more prevalent in Sydney during the colder months of the year. Sixty cases occurred in the cold period, as compared with forty-one in the warm period. Furthermore, the disease appears to be more virulent in the cold months, as the mortality rate is decidedly higher—10% as against 7·3%.

It is difficult to say why this apparent increase in severity in the colder months should occur. Possibly it may be associated with dietary factors and increased food intake, and possibly diminished intake of fluid.

TABLE IV.

Showing number of cases occurring in each month at the Women's Hospital.

Year.	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
1924	2	1	1	–	–	2	3	3	–	–	–	1	13
1925	–	–	1	1	–	1	1	1	1	2	–	–	8
1926	1	–	1	1	–	–	1	–	1	1	3	1	10
1927	1	–	–	–	1	–	–	4	–	3	1	1	11
1928	1	1	–	1	–	3	–	–	–	1	1	–	8
1929	–	–	4	2	–	1	1	1	–	–	–	–	9
1930	–	3	–	1	3	1	–	3	3	–	1	2	17
1931	–	1	2	1	2	–	2	–	4	–	–	–	12
1932	–	–	–	–	1	–	2	3	–	1	1	–	8
1933	–	1	2	1	–	1	–	–	–	–	–	–	5
Total	5	7	11	8	7	9	10	15	9	8	7	5	101

Williams mentions that Harrar, of New York, over ten years found an increased incidence in early spring, and he concludes that the increase is due to the cold, damp weather prevailing at that time. He says that it is generally accepted that eclampsia occurs less commonly in warm climates and in people whose diet is largely vegetable.

The age at which eclampsia occurred in the Women's Hospital series is shown in Table VI.

TABLE VI.

Age in Years.	Number of Cases.	Group Totals.
14	1	
15	2	
16	4	
17	8	44
18	9	
19	12	
20	8	
21	5	
22	5	
23	4	28
24	5	
25	2	
26 to 30	7	
31 to 35	10	20
36 to 39	10	
40 to 45	5	5
Over 45	1	1

Between the ages of fourteen and twenty years there were 44 cases with no deaths; between 21 and 30 there were 28 cases with two deaths; between 31 and 39 there were 20 cases with 4 deaths; between 40 and 45 there were 5 cases with no deaths. Over the age of 45 there was 1 case with no death. There were three cases in which the age was not mentioned, and the three patients died. The youngest patient was 14 years old; the eldest was aged 47 years.

The parity of the patients is shown in Table VII. It will be seen that 68 were *primigravidæ*, 31 were *multiparæ*; in two cases no information as to parity was available.

TABLE VII.

Parity.	Number of Cases.
1	68
2	11
3	5
4	5
5	3
6	3
7	2
9	1
12	1

Ætiology.

There are many theories of the cause of eclampsia, but I do not propose to discuss these. Eclampsia is usually preceded by albuminuria of pregnancy or preeclamptic toxæmia, and by a study of this condition the cause of eclampsia will probably be found. The theory at present most widely held is that the cause is some toxic body elaborated by the placental chorionic epithelium. Biochemical studies may eventually elucidate the problem, but although they are of value, chief reliance at present must be placed upon the clinical features.

I believe that by a careful study of past illnesses information can be obtained which is of great value in warning us that certain patients are liable to the occurrence of eclampsia. The illnesses which are to be specially inquired for are: general infections, scarlet fever, rheumatic fever, diphtheria, pneumonia, influenza, typhoid fever, tonsillitis, particularly recurring tonsillitis, quinsy, oral sepsis and sepsis in the upper part of the respiratory tract, and chronic sepsis in any part of the body. I have found that when one or more of these diseases has been present, albuminuria almost invariably appears during the last weeks of gestation. Recurring tonsillitis has been shown to be due in 90% of cases to streptococcal organisms, and I should like to stress the need for specifically inquiring for this, because when this is associated with other infections some kidney damage is very likely to be present which is unrecognizable by ordinary tests, but which shows itself as albuminuria in the latter part of pregnancy. In confirmation of this belief, I was interested to read in the tenth (1930) edition of "A Short Practice of Midwifery", by Jellett, the following remarks on the ætiology of eclampsia:

> To the foregoing causes I may add a fourth condition, the actual importance of which has not as yet been fully assessed.
>
> 4. Disease occurring in previous pregnancies, particularly when chronic organic defects have resulted therefrom.
>
> Lawrance, who has investigated a series of cases from this standpoint, came to the conclusion that there was a direct causal relation between such diseases and eclampsia, that the most important diseases were general infections, influenza, pneumonia, acute rheumatism, scarlet fever, furunculosis, and typhoid; and the most important defects those of the intestinal organs, the kidneys, and the heart. On the other hand, he was unable to trace any connection between eclampsia and diseases occurring during the same pregnancy.

This aspect of eclampsia and preeclamptic toxæmia is one to which, I think, greater attention should be paid in the future. In the histories of the present series this was not inquired into very fully. In 50 cases no mention of previous illnesses was made. In 23 cases previous illnesses were put down as "nil". In 28 cases only were previous illnesses found and noted.

In my experience the words "previous illnesses nil" do not give an accurate picture of the past life of the patient. The diseases mentioned above should be specially inquired for as a routine measure in every case. Patients in their ignorance regard tonsillitis, quinsy, oral sepsis *et cetera* as being of such minor importance that they do not mention them. I have repeatedly found in preeclamptic toxæmia that some of these diseases have been present when a careful inquiry has been made, in spite of the fact that "previous illnesses nil" had been previously recorded.

In the 28 cases available for study the following diseases had occurred:

Pneumonia	5 cases
Diphtheria	4 cases
Frequent sore throats	4 cases
Appendicectomy	4 cases
Scarlet fever	3 cases
Oral sepsis	3 cases
Rheumatic fever	2 cases
Chronic mastoid disease	2 cases

Other diseases noted once included chorea, *erythema multiforme exudativa*, influenza, infantile paralysis, typhoid fever, whooping cough, epilepsy, jaundice, bilious attacks. Measles was noted four times and doubtful diabetes twice.

Twin Pregnancy.

The liability to eclampsia is greater in twin pregnancy than in single pregnancy. In the British series it was found in three centres as follows: In London the proportion of twin pregnancies was 9·0%; in Edinburgh the proportion was 3·3%; in the north of England it was 2·8%. A total of 1,524 cases gave a mean proportion of 4·7%. In the Women's Hospital series there were four twin pregnancies in 101 cases. The usual frequency of twin pregnancy is 1 in 80.

Danger Signals.

The following seven phenomena are signs of danger: coma, a pulse rate over 120 per minute, a temperature above 39·4° C. (103° F.), a number of fits greater than ten, a urine which becomes solid on boiling, the absence of œdema, a systolic blood pressure of over 200 millimetres of mercury.

It is obvious that in a disease which presents such a varying degree of severity it is very useful to have some method of distinguishing mild from severe cases. The London committee, from a study of their cases, were led to the conclusion that the above phenomena were signs of danger, and when a patient exhibited any two of these the case was classified as severe; if less than two of the phenomena were present, as mild. I have classified the cases in this series along similar lines.

Table VIII shows the maternal mortality in the British series and in the series reported by Stroganoff, and from Williams in America.

TABLE VIII.

Series.	Cases.	Percentage Maternal Mortality.
London	547	21·9
Edinburgh	148	25·0
Dublin	204	10·29
North of England	804	24·43
Midland	302	25·10
Williams (average American)		20·0–25·0
Stroganoff	5,797	10·0
Women's Hospital, Crown St.	101	8·9

It will be seen from the above table that the Women's Hospital has the lowest maternal mortality rate. In fairness, however, to the British series, it must be remembered that all their figures were taken from a series of hospitals in each centre, and only the average rate was given. The Dublin figures were the result of a standardized treatment; Stroganoff's figures were made up of a series treated by various practitioners following the prophylactic methods laid down by him. It is usually stated that the average mortality rate is about 20% in Great Britain.

Stroganoff asserts that the maternal mortality rate can be lowered still further, and he has quoted a series of 300 eclampsias with a maternal mortality of only 2·6%; and he says that even this small percentage can really be made non-existent if his prophylactic methods are carefully carried out. His book, "The Improved Prophylactic Method in the Treatment of Eclampsia", contains a full description of his methods and is a most valuable contribution to the study of this disease.

In the present series nine deaths occurred. The details of the fatal cases are as follows.

T., a *primigravida*, was admitted to hospital on July 26, 1926, and died in her eighth fit, three and three-quarter hours after admission. The duration of pregnancy was not stated. She was admitted in deep coma and her urine was solid with albumin on boiling. The systolic blood pressure was 190 and the diastolic pressure 104 millimetres of mercury, the pulse rate was 104, the temperature was 35·6° C. (96° F.), and she had general œdema.

E.I., twenty-eight weeks pregnant, was admitted to hospital on January 2, 1924, and died nine hours after admission. She had had three fits before admission. She was deeply comatose and her urine was solid on boiling.

E.D., who had had four pregnancies, was admitted to hospital on March 11, 1925, and died twelve hours after admission. She was thirty-six years of age and was thirty-five weeks pregnant. The urine was solid with albumin on boiling. The patient was in deep coma. The temperature was 39·3° C. (102·8° F.); the pulse rate was 148. The cervix was dilated, the membranes were ruptured, and gauze was packed into the cervix. The patient had the usual treatment of morphine and stomach and bowel wash-out.

E.T., a *primigravida*, aged twenty-nine, thirty-four weeks pregnant, died one day after admission to hospital. She had deep coma, "solid albumin", general œdema, a pulse rate of 120, a systolic blood pressure of 200 and a diastolic pressure of 125 millimetres of mercury. She had eighteen *ante partum* fits. She gave a history of frequent sore throats, whooping cough, infantile paralysis and measles. An attempt was made to induce labour with gauze in the cervix, but she died undelivered. She had the usual morphine and eliminative treatment.

G.C. was admitted to hospital on August 2, 1924, with deep coma and urine that became solid with albumin on boiling. She was very cyanosed. Her temperature was 38·9° C. (102° F.), her pulse rate was 150, and she had only two fits. Her history was very incomplete. She had morphine and eliminative treatment with saline solution given intravenously, but she did not respond. She died twenty-nine hours after admission.

B.S., a patient with five children, aged thirty-seven, and thirty-nine weeks pregnant, was admitted to hospital on August 11, 1927. Her temperature was 37·8° C. (100° F.), her pulse rate was 160. Her systolic blood pressure was 144 and her diastolic pressure 100 millimetres of mercury. She had ten *post partum* fits and slight œdema. She was in deep coma and had "solid albumin". She was admitted in the second stage of labour of a twin pregnancy, in which a foot was presenting. This foot belonged to the second twin. The first twin was a full breech with intact membranes. The foot was pushed up. The membranes of the first twin were ruptured and the first twin was delivered. It had a *spina bifida*. The second twin was then delivered. There were separate placentæ with numerous succenturiate lobes. An intrauterine douche was given at the end of delivery. The patient slept for two hours, then she woke with pains under the left breast and vomiting.

She had a fit seven hours after delivery, and had ten fits altogether, with deep coma. She had icteric tinged conjunctivæ, and blood and bile in her urine. She died twenty-four hours after delivery.

E.U., with seven children, and aged thirty-three years, thirty-four weeks pregnant, was admitted to hospital on August 22, 1930, and delivered on the same day of a still-born male child weighing four pounds ten ounces. She had nineteen fits. She was deeply comatose on admission and for three days afterwards. She was semi-comatose for a further two days, and her urine was diminished to about one and a half to six ounces a day. The day after admission her blood urea was 81 milligrammes *per centum*, the blood creatinin was 3·5 *per centum*, and the blood cholesterol 400. Granular and hyaline casts were present in the urine. She had the usual treatment. Her blood urea rose to 204 milligrammes, and the blood creatinin to 11 milligrammes *per centum*, and she died ten days after admission. The previous history was that she had had swelling of the hands and face two nights before admission.

F.M.F., a *primigravida*, aged thirty-seven, was admitted to hospital on September 5, 1931, at term. She was in labour for 101 hours 50 minutes. She had early rupture of the membranes, and finally dilatation had to be completed manually and the forceps applied. The position was left occipito-anterior and a still-born female child, eight pounds in weight, was delivered. The perineum was ruptured. She had three *ante partum* fits. The systolic blood pressure was 170 and the diastolic pressure 90 millimetres of mercury. The pulse rate was 116. The patient was in deep coma. The temperature was normal. The urine became solid with albumin on boiling. She developed general peritonitis in the puerperium and died on the nineteenth day after admission. Previous illnesses were a mastoid operation and removal of all her teeth.

O.M.A., a *primigravida*, aged twenty-four, was admitted to hospital on August 3, 1924. She was about thirty weeks pregnant. She had complained of dimness of vision and œdema of the face for two weeks before admission. She had been treated by a private doctor. She was admitted deeply comatose, with "solid albumin" in the urine. The systolic blood pressure was 120 and the diastolic pressure 85 millimetres of mercury. She had three fits before admission, and her urine contained hyaline and granular casts. She was comatose for three days after admission and the urine was scanty and solid on boiling for four days after admission. She improved slightly under treatment, but nine days after admission she had another fit; her systolic blood pressure rose to 185 and her diastolic pressure to 120 millimetres of mercury. The urine again became scanty and contained pus. Blood urea was 101 milligrammes *per centum*. The urea nitrogen was 47 milligrammes *per centum*. Surgical induction of labour was performed by packing gauze into the cervix. She had a prolonged labour and was delivered of a still-born female child of two pounds four ounces in weight. The pains were weak. She had two *post partum* fits and again had "solid albumin" and deep coma, and finally her respiration failed and she died two days after delivery and twenty-three days after admission.

Table IX shows the classification into mild and severe cases of London, Midland and the Women's Hospital series. In the other reports this classification was not adopted. The figures of the Midland series were about the same as in this series, but the proportion of severe cases was much greater than that of London.

TABLE IX.
Classification.

Series.	Mild.	Severe.
London	62·0%	38·0%
Midland	47·4%	52·6%
Women's Hospital, Crown Street	48·5%	51·4%

Table X shows the mortality rates of the three previous centres. In our series no deaths occurred in the mild variety, whereas both the others show a 6% death rate. In the severe variety considerable difference is shown. Eclampsia in the Midlands evidently seems to be more severe than in London, because proportion of severe cases and the death rates are higher than in London, but it is pleasing to note that our death rate in the severe variety compares more favourably than either, being almost half that of London, although our proportion of severe cases was much greater.

TABLE X.
Mortality rates, mild and severe.

Series.	Mild.	Severe.
London	6·4%	32·4%
Midland	6·7%	42·6%
Women's Hospital, Crown Street	0·0%	17·3%

Table XI shows the incidence of *primigravidæ* and *multiparæ*, and Table XII shows the percentage mortality in *primigravidæ* and *multiparæ*. It is interesting to see how the incidence between *primigravidæ* and *multiparæ* is so similar in all the centres and also in the Women's Hospital series. The percentage mortality also in the British series is almost the same in the various centres and shows the slightly higher rate in *multiparæ*. The mortality rate in *multiparæ* at Crown Street, and also in *primigravidæ*, is much less than in the British series, but again shows the same increased rate for *multiparæ*.

TABLE XI.
Showing incidence in primigravidæ and multiparæ.

Series.	Cases.	*Primigravidæ.*	*Multiparæ.*
London	488	69·8%	30·2%
Edinburgh	212	75·0%	25·0%
Dublin	204	67·15%	29·3%
North of England	804	70·7%	29·3%
Midland	302	67·15%	32·85%
Women's Hospital, Crown Street	99	68·5%	31·3%

TABLE XII.
Showing the percentage mortality in primigravidæ and multiparæ.

Series.	Cases.	*Primigravidæ.*	*Multiparæ.*
London	488	23·4%	27·0%
Midland	302	23·8%	27·5%
North of England	804	20·6%	27·8%
Women's Hospital, Crown Street	99	5·8%	9·9%

Pregnancy is the best test we have in assessing the functional powers of the organs of the female body. Any acquired or inherent weaknesses usually are manifested some time or other during gestation. Therefore it is not surprising to find eclampsia so prevalent in *primigravidæ*. Table VII shows this very well. There we see that 68 cases occurred in the first pregnancy, 11 cases in the second, 5 each in the third and fourth, 3 in the fifth and sixth, 2 in the seventh, 1 in the ninth, and 1 in the twelfth.

After the first pregnancy there is a steady and continuous drop in the frequency, but it is interesting to see that one case occurred in the twelfth pregnancy, thus proving again the necessity for antenatal supervision in every pregnancy. It does not matter how many children previously may have been born normally, the risk of eclampsia developing is always present and should be guarded against.

Table VI shows the age at which eclampsia developed in the Crown street series. Text books give twenty-four years as the average age at which eclampsia develops. In this series 44 cases occurred between the ages of fourteen and twenty years, nearly half the number of cases, but no deaths occurred. Twenty-eight cases occurred between the ages of twenty-one and thirty with two deaths. Twenty cases occurred between the ages of thirty-one and thirty-nine years with four deaths. Five cases occurred between the ages of forty and forty-five years, and one patient was forty-seven years old. These facts are interesting as showing again that although eclampsia occurs so frequently among young persons, the mortality rate is low with them, but increases considerably over the age of thirty.

TABLE XIIIA.

Group.	Mild.	Severe.	Total
Primigravidæ	33	35	68
Multiparæ	16	15	31

Nine deaths occurred, but in two the parity was not noted; all were of the severe type.

TABLE XIIIB.

Group.	Severe.	Number of Deaths.	Mortality Rate.
Primigravidæ	35	4	11·4%
Multiparæ	15	3	20·0%

I thought it would be interesting to analyse these figures still further, and Tables XIIIA and XIIIB show that although eclampsia occurred about twice as frequently in *primigravidæ* as in *multiparæ*, the proportion of mild to severe in the two was about the same. As there were no deaths in the mild variety, it would seem that the prognosis in both *primigravidæ* and *multiparæ* in the mild type is about the same, but in the severe type the prognosis is twice as bad in the *multiparæ*.

The percentage of infants that survived and left hospital alive is shown in Table XIV. This shows that Dublin had the best results, closely followed by Crown Street, both being considerably better than all of the others.

TABLE XIV.

Percentage of infants that survived.

Series.	Total Number of Infants.	Survived.	Percentage.
London	448	242	54·0
Edinburgh	207	84	44·4
Dublin	204	134	65·6
Midland	133	60	52·0
North of England	Not given.	Not given.	43·4
Women's Hospital, Crown Street..	105	67	63·8

There were four twin pregnancies, so 105 infants were available for study. Thirty-eight infants failed to survive, including two of the twins.

The fatal results were as follows:

Macerated	4
Still-born	19
Died within two hours after birth	1
Died within four hours after birth	1
Died within ten hours after birth	1
Died within twenty hours after birth	1
Died within twenty-four hours after birth ..	2
Died seven days after birth	1
Died twelve days after birth	1
Undelivered	5

The fœtal mortality was 36%.

Williams states that a fœtal death rate of approximately 50% is not surprising, and in his clinic but little more than one-third of the children left the hospital with their mothers.

Stroganoff states that with his improved prophylactic method there is a 10% mortality of children, so it is evident that with improved methods a much better percentage of recoveries should be looked for in the future.

TABLE XV.

Showing Period of Gestation at which Eclampsia Supervened. Total number of British cases, 546; Women's Hospital, 95, giving the following mortality rates.

Series.	Before Thirty-Sixth Week.	Thirty-Six Weeks and After.
London	22·2%	17·9%
Midland	31·8%	21·8%
Women's Hospital, Crown Street	17·2%	3·03%

Sixty-seven *primigravidæ* and twenty-eight *multiparæ* were available. Details were not noted in six cases. The British figures do not show a very great difference between the mortality rates in cases occurring before the thirty-sixth week and after the thirty-sixth week, the mean of the two centres giving 24·0% before and 19·5% after. The Crown Street results differ considerably from this and show that eclampsia occurring before the thirty-sixth week is associated with about a six times greater mortality rate.

Table XVI shows this very clearly. This table shows that not many cases occur between the twenty-eighth and thirty-second weeks, but when eclampsia does occur, the severe variety predominates in both *primigravidæ* and *multiparæ* in the proportion of two to one, and is associated with a mortality of 16·6%. Between the thirty-second and thirty-sixth weeks more cases occur with a greater preponderance still of the severe variety in both types of patients, particularly in the *primigravidæ*, and associated with a higher mortality of 17·6%. Between the thirty-sixth week and the fortieth week the greater number of cases occur with a marked preponderance of mild cases in both classes of patients, especially in *multiparæ*, and a very much lower mortality rate of 3·03%. At term the number of cases was about half the former group; the mild variety predominates, but more

TABLE XVI.
(*Women's Hospital Series.*)

Duration of Pregnancy.	*Primigravidæ.*		*Multiparæ.*		Total.	Died.	Mortality Rate.
	Mild.	Severe.	Mild.	Severe.			
Between 28 and 32 weeks	2	4	2	4	12	2	16·6%
Between 32 and 36 weeks	1	9	2	5	17	3	17·6%
Between 36 and 40 weeks and over	31	20	11	4	66	2	3·03%
At Term	16	11	9	1	37	1	2·7%

particularly in *multiparæ*, and the mortality rate is still lower, 2·7%. This supports the belief that eclampsia is more severe if it occurs in or before the thirty-sixth week and is associated with a definitely higher mortality.

Coma.

The London Committee divided coma into three groups, as follows: (i) Deep coma from which the patient cannot be roused. (ii) Coma in which a stimulus, such as a needle prick, will evoke some sort of response. (iii) Drowsiness in which the patient can be made to speak by attempts to waken her. It was not an easy matter to determine accurately the degree of coma in the series reviewed. No mention of coma was made in 30 cases. These, as it was impossible to know whether coma was present or absent, I have omitted altogether. Of the remaining 71, 31 were classified as deep coma, 19 as coma, and 21 as drowsiness.

TABLE XVII.

Degree of Coma.	Number of Cases.	Incidence.	Deaths.	Percentage Mortality.
Deep coma—				
London	60	22·9%	38	63·4
Women's Hospital, Crown Street	31	43·6%	9	29·03
Coma—				
London	93	33·5%	19	20·4
Women's Hospital, Crown Street	19	26·7%	0	0·0
Drowsiness—				
London	109	41·6%	6	5·4
Women's Hospital, Crown Street	21	29·5%	0	0·0

Table XVII demonstrates that deep coma was associated in both the series with a very high mortality rate. In a series of 718 cases from London, Edinburgh and Midland, the mean mortality rate was: no coma, 5·7%; coma, 20·0%; deep coma, 54·0%. The degree of coma thus is very important in prognosis.

Pulse Rate.

The London Committee considered the pulse rate under three headings: (i) over 120, (ii) 120 to 90, (iii) below 90. The pulse rate according to these groups in the Women's Hospital series is set out in Table XVIII.

TABLE XVIII.

Pulse Rate.	Cases.	Died.	Mortality.	London Mortality.
Over 120	36	4	11·1%	40·0%
120 to 90	61	3	4·9%	9·4%
Below 90	2	0	0·0%	5·4%

In two cases the pulse was not recorded. Nine patients had a pulse of 150 and over. The highest was 180. A pulse rate of over 120 is of bad prognostic significance.

Onset of Fits in Relation to Labour.

In regard to the onset of fits in relation to labour, the mean results from London, Edinburgh and Midland in 899 cases were: onset before labour, 61·5%; onset during labour, 19·2%; onset after labour, 19·3%. The London series and Women's Hospital series mortality rates are compared in Table XIX.

TABLE XIX.

Time of Onset.	London Series.			Women's Hospital Series.
	Number of Cases.	Deaths.	Mortality Rate.	Mortality Rate.
Before labour	287	59	20·5%	18·4%
During labour	84	14	16·6%	0·0%
After labour	76	21	27·6%	5·0%

The times at which fits occurred in the Women's Hospital series are shown in Table XX.

TABLE XX.

Fits.	*Primigravidæ.*		*Multiparæ.*		Total.	Deaths.	Mortality Rate.
	Mild.	Severe.	Mild.	Severe.			
Ante partum	7	18	4	9	38	7	18·4%
Intra partum	6	8	3	1	18	0	0·0%
Post partum	10	2	5	3	20	1	5·0%
Ante and *intra partum*	4	4	0	1	9	0	0·0%
Ante and *post partum*	0	1	0	0	1	1	100·0%
Intra and *post partum*	4	2	4	0	10	0	0·0%
Ante, intra and *post partum*	0	0	0	0	0	0	0·0%

In the Women's Hospital series only 96 cases were available for study. Details were not mentioned in five cases. Table XX shows that in the great majority of cases fits start before the onset of labour, and that the number starting during or after labour are about equal. The mortality rate varies in different series. The London series showed the highest rate in the *post partum* fits. In other centres the *ante partum* rate was the highest. I had always been of the opinion that the earlier in pregnancy the fits started, the worse the prognosis, but that at the same time the *post partum* variety was associated with a high mortality; and so I was rather surprised to find that in this series the *post partum* mortality rate was as low as 5%, as compared with 18·4% in the *ante partum* variety. This table also shows the increased severity of the cases with the *ante partum* variety.

The average number of fits *per capita* in the 1,051 British cases and the 98 Women's Hospital cases are shown in Table XXI.

TABLE XXI.

Centre.	Recovered.	Died.
London	6·8	12·7
Edinburgh	6·1	10·0
Dublin	6·4	10·0
Midland	7·3	14·5
Women's Hospital, Crown Street	5·2	10·1

These figures are very similar, but go to show that in the Midlands the variety of eclampsia seems to be very severe. Sixteen patients in this series had 10 or more fits; the highest number was 19. Twelve were *primigravidæ*, four were *multiparæ*. But whereas only one *primipara* died, three out of the four *multiparæ* died, which shows that a number of ten fits or more is much more serious in a *multipara* than in a *primipara*. The number of fits in the nine patients who died varied from two to nineteen. In three cases the number was not recorded.

Temperature.

The London Committee divided the patients according to their temperatures into the three groups as follows: Group I, above 39·4° C. (103° F.); Group II, 37·8° to 39·4° C. (100° to 103° F.); Group III, below 37·8° C. (100° F.). The temperatures in the London series and in the Women's Hospital series are compared in Table XXII.

TABLE XXII.

Group.	Number of Cases.	Deaths.	Mortality.
Above 39·4° C. (103·0° F.)—			
London	39	29	74·3%
Women's Hospital, Crown Street	0	0	0·0%
37·8° C. to 39·4° C. (100·0° to 103·0° F.)—			
London	125	14	11·2%
Women's Hospital	50	5	10·0%
Below 37·8° C. (100·0° F.)—			
London	187	11	5·9%
Women's Hospital	48	2	4·1%

Table XXII shows that the mortality rates in cases of temperature above 39·4° C. (103° F.) in London was very high. In the Women's Hospital series only two patients had a temperature of 39·4° C. (103° F.) and none died. Nine patients had a temperature between 38·9° and 39·4° C. (102° and 103° F.), and of these two died, giving a mortality rate of 22·2%. Apart from the first group, the two series correspond very closely. In the fatal cases in the Women's Hospital series the temperatures recorded were 35·6° C. (96° F.), 36·9° C. (98·4° F.), 37·8° C. (100° F.), 38·0° C. (100·4° F.), 38·3° C. (101° F.), 38·9° C. (102° F.), 39·3° C. (102·8° F.). In two cases the temperature was not recorded.

Blood Pressure.

The blood pressure (systolic) and mortality rate in the two series are set out in Table XXIII.

TABLE XXIII.

Series.	Below 140 Millimetres.	140 to 200 Millimetres.	Over 200 Millimetres.
London	21·0%	20·0%	36·0%
Edinburgh	18·5%	18·5%	35·3%
Women's Hospital, Crown Street	0·0%	8·5%	0·0%

The London Committee considered the blood pressure from the three headings, below 140 millimetres of mercury, 140 to 200 millimetres, and over 200 millimetres. As is seen, the mortality figures of the Women's Hospital differ considerably from the London and Edinburgh series.

The blood pressures (systolic) in the Women's Hospital series are set out in Table XXIV.

TABLE XXIV.

Height of Blood Pressure in Millimetres of Mercury.	Cases.	Deaths.	Mortality.
Below 140	10	0	0·0%
140 to 200	70	6	8·5%
Over 200	12	0	0·0%

Table XXIII shows that in London and in Edinburgh there was not much difference in the mortality rate when the blood pressure was below 140 millimetres and 140 to 200 millimetres, but was definitely increased when the blood pressure rose to over 200 millimetres. In the Women's Hospital series the blood pressure was not recorded in nine cases; in the seven fatal cases in which it was recorded the blood pressure was as follows: 144, 170, 185, 190, 194, 200 millimetres of mercury.

Amount of Albumin.

In the Women's Hospital series, in 61 cases the urine became solid on boiling. Nine of these patients died, giving a mortality rate of 14·9%. As there were no deaths with a less amount, I did not work out the numbers with a less amount.

The London Committee divided their cases into three groups: Group I, small amount (cloud); Group II, large amount (deposit); Group III, solid on boiling. Their results were as shown in Table XXV.

TABLE XXV.

Amount of Albumin.	Number of Cases.	Deaths.	Mortality.
None	2	0	0·0%
Small amount (cloud)	38	30	14·0%
Large amount (deposit)	213	30	14·0%
Solid on boiling	130	33	25·3%

At the Women's Hospital we usually estimate the amount of urine as solid, three-quarters, one-half, one-quarter, one-sixth, one-eighth, or as a cloud or a trace, as the case may be.

Œdema.

The London Committee stated that it is impossible to describe any accurate standard of œdema, but they grouped it into three clinical classes, according to whether there was "none", "slight", or "great and universal". They found that œdema was a favourable sign, while among those patients who had no œdema the mortality was nearly twice as great. The mortality rates were respectively 16% and 29%. In the Women's Hospital series I found it very difficult to decide the amount of œdema present, and so thought that no good would result from an imperfect analysis. I have always held the opinion expressed by the London Committee.

Table XXVI shows the mortality rates in the various seven warning phenomena, and it will be seen that in this series deep coma is the most dangerous, closely followed by ten fits and over. Then comes the temperature, amount of albumin, pulse rate, and finally the blood pressure. The amount of œdema was not determined.

TABLE XXVI.
(Women's Hospital Series.)

Phenomenon.	Mortality Rate.
Deep coma	29·03%
Number of fits ten and over	25·0%
Temperature 38·9° C. (102·0° F.) and over	18·1%
Solid albumin	14·9%
Pulse rate over 120	11·1%
Blood pressure 140 to 200 millimetres of mercury ..	8·5%

Recurrent Toxæmia.

In recent years attention has been directed to the large proportion of cases showing a recurrence of toxæmia in subsequent pregnancies, and the incidence of chronic nephritis resulting from eclampsia and preeclampsia. Young, of Edinburgh, reports a 3% incidence of chronic nephritis after eclampsia and an 8% incidence after preeclampsia. He also reports a recurrence of toxæmia in subsequent pregnancies of 55·8%. Donovan reported albuminuria on discharge after eclampsia in 19·3% of *primiparæ* and in 54·5% of *multiparæ* who had had toxæmia during previous pregnancies. Time forbids a discussion of these interesting facts. However, in the present series three patients had a recurrence of eclampsia as follows. A patient, who had had five children, had eclampsia twice in this series, on March 20, 1929, and on May 3, 1930. A patient, in her fourth pregnancy, had fits at the eighth month with her two previous pregnancies. A patient, in her third pregnancy, had eclampsia with her first pregnancy.

One patient stated that her mother had had eclampsia when she (the patient) was born. Persistence of albumin in the urine may be regarded as some evidence of chronic renal damage with liability to recurrence of toxæmia in subsequent pregnancies, and it is interesting to note that of the *primiparæ* 20·7% and of the *multiparæ* 30·7% had albumin still in their urine on discharge.

Preventive Treatment.

Cameron, of Glasgow, has introduced a treatment by alkalis and calcium which gives very promising results. A tablet containing 0·42 gramme (seven and a half grains) of calcium soda lactate, 2·4 grammes (forty grains) of potassium citrate, and 1·2 grammes (twenty grains) of sodium bicarbonate, which is very easily soluble in water, is given three or four times a day to all albuminuric patients except those whose albuminuria is due to chronic nephritis. In severe cases of albuminuria a sterile solution of calcium gluconate, made up in ampoules of ten cubic centimetres, is administered intravenously. We have been trying this lately at the Women's Hospital with dramatic results in some cases of toxæmia.

Stroganoff's Treatment.

Stroganoff's treatment is as follows.

Beginning of treatment: Morphine, 0·015 gramme (one-quarter of a grain), is given. The patient is under light chloroform anæsthesia.

In one hour's time after beginning of treatment: Chloral hydrate, 1·92 grammes (thirty-two grains), is given to conscious patients *per os* with milk; to unconscious patients it is given per rectum under chloroform anæsthesia with milk and saline solution, ää 105 cubic centimetres (three and a half ounces).

In three hours' time after beginning treatment: Morphine, 0·015 gramme (one-quarter of a grain), is given. The patient is under light chloroform narcosis (20 to 80 drops).

In seven hours' time after beginning of treatment: Chloral hydrate, 1·92 grammes (thirty-two grains), is given as before.

In thirteen hours' time after beginning of treatment: Chloral hydrate, 1·44 grammes (twenty-four grains). Chloroform anæsthesia is not used if no fits have occurred during eight to ten hours.

In twenty-one hours after beginning of treatment: Chloral hydrate, 1·44 grammes (twenty-four grains), is given as before.

Thus morphine and chloral hydrate are given at stated intervals until the fits are controlled. Venesection is used and 450 cubic centimetres (fifteen ounces) of blood are withdrawn if the fits or coma persist. The bag of membranes is ruptured if the patient is in labour, and the second stage of labour is terminated with forceps. The patient is isolated in a quiet dark room. The air passages are cleansed after the fits. Fresh air and oxygen are given if the patient is cyanosed; cardiac stimulants are administered if necessary.

TABLE XXVII.

Method of Delivery.	London.		Midland.		Aggregate.		Women's Hospital, All Cases Severe.
	Mild.	Severe.	Mild.	Severe.	Mild.	Severe.	
Natural delivery	6·0%	20·5%	3·6%	44·0%	4·5%	36·9%	1·8%
Assisted delivery	5·0%	33·3%	8·0%	29·4%	5·6%	31·7%	7·1%
Induction	5·1%	20·8%	11·7%	40·0%	6·6%	26·4%	18·7%
Cæsarean section	9·8%	43·2%	50·0%	75·0%	11·3%	46·3%	None done
Accouchement forcé	25·0%	60·0%	14·3%	66·0%	18·1%	63·1%	None done

Method of Delivery and Maternal Mortality Rate.

In Table XXVII the maternal mortality rate in the several centres is shown in relation to the method of delivery.

The method of delivery adopted in the Women's Hospital series is shown in Table XXVIII.

TABLE XXVIII.

Method of Delivery.	Cases.	Deaths. (Severe Cases.)	Mortality Rate.
Natural delivery	54	1	1·8%
Induction of labour and spontaneous delivery	16	3	18·7%
Assisted delivery (forceps, induction and forceps, manual manipulation)	28	2	7·1%
Undelivered, no induction	3	3	100·0%

From Table XXVII it is seen that in the British series natural delivery, assisted delivery and induction gave the best results, in the order mentioned, in the mild variety, whereas the order is exactly reversed in the severe variety. The mortality rate from Cæsarean section was almost twice as great in the mild variety, and very large in the severe also. *Accouchement forcé* gave very bad results in both varieties and should be abandoned.

In the Women's Hospital series there were no deaths in the mild variety; all were in the severe group; and it is seen that natural delivery gave by far the best results. Assisted delivery came next, because the majority of these occurred in low forceps cases near term. Induction gave the worst results, because this was done in most cases before the thirty-sixth week of pregnancy in patients who were not responding to treatment. Cæsarean section was not performed, and in view of the bad results shown by the British figures and by statistics published in other parts of the world, it should be reserved for patients with purely obstetric indications, apart altogether from the eclampsia. Three patients died undelivered; details of these cases have been considered.

General Treatment.

General treatment was governed by three main principles: (i) the control of the fits by sedatives, (ii) the elimination of the toxins, (iii) supporting the patient's strength by general nursing measures.

The Control of the Fits by Sedatives.

On admission, if the patient was suitable, morphine, 0·015 to 0·03 gramme (one-quarter to one-half grain), was at once administered; the dose depended upon the condition of the patient. This is repeated in doses of 0·008 to 0·011 gramme (one-eighth or one-sixth of a grain) at varying intervals, depending upon the persistence of the fits, sometimes every hour. Chloral hydrate, 0·6 to 1·5 gramme (ten to twenty-five grains), and potassium bromide, 1·8 to 2·4 grammes (thirty to forty grains), are given if the patient is very restless. These are given by mouth if the patient can swallow, or by stomach tube or by rectum if the patient is unable to swallow. Paraldehyde, 24 cubic centimetres (six fluid drachms), is given *per rectum* if the respirations become too slow after morphine.

Eliminative Treatment.

Bowel lavage is given after the morphine on admission. The patient is put on her left side and two pints of saline solution or bicarbonate of soda solution are run into the large bowel and siphoned out, and this is repeated until a good fæcal result has been obtained. This is usually done under light chloroform anæsthesia. This may take half an hour or longer. It is repeated every twelve hours as a rule, but in some cases at more frequent intervals if good results have not been obtained previously. Stomach lavage is done in certain cases when there is much vomiting or when it is desired to get aperients into a comatose patient. It is done under light chloroform anæsthesia.

Compound powder of jalap and magnesium sulphate are left in after the stomach has been washed out when the patient is unable to swallow. Compound powder of jalap, 4·0 grammes (two drachms), is administered at night, followed in the morning by magnesium sulphate, 15 to 30 grammes (half to one ounce) in certain cases at six-hourly intervals.

General Nursing Measures.

Patients are put on water only at first, and if improvement is taking place, on fruit juice, greens, bread and butter, milk foods *et cetera*.

The air passages are carefully attended to and the patients are kept on their side to allow the mucus to escape. Oxygen is given if there is any cyanosis. Digitalin is given if any cardiac stimulation is indicated. Veratrone, 0·18 to 0·3 mil (three to five minims), is given if the blood pressure is unduly high. Venesection was done in a few cases. Glucose and saline solution with or without insulin was given intravenously in a few cases.

These were the lines along which treatment was carried out in the cases in this series. There were

individual variations in many cases, depending upon the various honorary surgeons in charge.

Conclusions.

As a result of a study of 101 cases of eclampsia treated by all members of the honorary medical staff at the Women's Hospital, Crown Street, Sydney, I have formed the following conclusions:

1. A symptom suggestive that eclampsia may occur is a blood pressure which is low in the early months of pregnancy, which gradually rises in the later months of pregnancy to 140 millimetres of mercury or over, and which is associated with œdema and albuminuria.

2. Eclampsia in Sydney is more prevalent in the winter months, and at that time the mortality rate is higher.

3. Eclampsia in Sydney is more prevalent in persons below the age of twenty-one years, but may occur at any age. The mortality is low before the age of twenty-one years and is higher over the age of thirty years.

4. The prognosis is worse in eclampsia supervening before the thirty-sixth week than later in pregnancy. The severe type predominates before the thirty-sixth week, and the mild predominates after the thirty-sixth week.

5. The classification adopted by the London Committee into mild and severe types is an advance in the study of the disease, and its general adoption would make future reports more valuable.

6. The incidence of eclampsia in Sydney is slightly lower than that of Great Britain, but the incidence in *primigravidæ* and *multiparæ* and the percentage of mild and severe cases are about the same.

7. The most important prognostic phenomena are: the degree of coma, the number of fits, the temperature, the amount of albumin, the pulse rate, and the height of the blood pressure in that order.

8. *Ante partum* eclampsia is associated in Sydney with a higher mortality than *post partum*.

9. The severe type of eclampsia is about twice as dangerous in *multiparæ* as in *primigravidæ*.

10. Eclampsia can be prevented in most cases by thorough ante-natal supervision, but not in all.

11. The history of previous illnesses is of value in determining the liability of eclampsia ensuing.

12. Eclampsia recurs in about 3% of cases and is followed by signs suggestive of some permanent renal damage in about 20% of cases.

13. Conservative treatment with a minimum of obstetric interference yields the best results.

14. The results of treatment at the Women's Hospital compare very favourably with the average results in Great Britain and America, but fall below Stroganoff's latest results.

15. Eclampsia shows no marked variation in its general manifestations in Sydney as compared with other parts of the world.

Reviews.

DIAGNOSIS AND TREATMENT OF TUBERCULOSIS.

OF Robert Koch's original pupils who studied the pathology and diagnosis of tuberculosis with him in Berlin in the ninth decade of last century but few remain to carry on his work. Of these the protagonist in England is Dr. Camac Wilkinson who, with magnificent courage, has reentered the fray in publishing a new book on tuberculin.[1] This small work consists of two parts. The first is on the pathology, diagnosis and treatment of chronic tuberculosis of the eye, such as *keratitis punctata*, scleritis, episcleritis, chorioiditis, iritis, iridocyclitis, *periphlebitis retinalis tuberculosa*, and possibly sympathetic ophthalmitis. All these lesions may, of course, have other causes—syphilis, gonorrhœa *et cetera*—but, as is shown by numerous cases, the chronic forms are often tuberculous. The common attribution of them to rheumatism is much less fashionable since Poncet and Leriche in France, Schaeffer in Germany, and Rolleston in England showed how often vague pains and inflammation of joints and other structures so frequently called rheumatism are really by-effects of an unrecognized tuberculosis. In a review of 53 such ocular cases, 43 of which, after their progress had rendered other causes unlikely, were diagnosed as tuberculous by reaction to tuberculin and successfully treated by hypodermic injection of tuberculin, and 10 of which did not react and formed useful controls, Dr. Wilkinson establishes his thesis that the possibilities of direct observation, aided, if necessary, by optical instruments, permit here a far clearer demonstration of the value of tuberculin than is possible in most other parts of the body. Dr. Charles Leonard Gimblett, an ophthalmic surgeon, who followed the progress of many of these patients, has been completely convinced, and in a foreword to the book he expresses his admiration for Dr. Wilkinson's profound knowledge, not only of tuberculosis, but also of general medicine and pathology, and his unqualified satisfaction with tuberculin as a diagnostic and therapeutic agent. Of the 43 cases referred to above, copious notes are given, and 31 of them are illustrated by charts.

The second part of the book is entitled "The Diagnosis, Treatment and Prevention of Phthisis". It contains descriptions of some new work and ideas, notably on the character and course of infection described by Ranke as the primary complex, but consists mostly of an eloquent exposition of the author's views, so often brilliantly stated, but, alas, so little appreciated, of the economic inadequacy of sanatorium treatment and of the best means for the prevention of acute tuberculosis in infants and children by the thorough exploitation of tuberculin at special clinics, the only means for the treatment of phthisis in tne aggregate available to a nation of limited monetary resources.

Camac Wilkinson's admirers, of whom there are many in the profession, though by no means so numerous and vociferous as his detractors, will receive this latest production with interest, gratitude and enthusiasm.

SCIENCE AND THE ECONOMIC SYSTEM.

DR. FRANK TRINCA, in his book, "Science and Democracy", shows that he sees in the present economic system two distinct relationships, that of science to industry and that of industry to finance.[2] Thus, modern industry is reacted upon by two separate forces, the net outcome being the series of booms and depressions with which we are so familiar.

[1] "Tuberculin: Its Vindication by Technique, with Special Reference to Tuberculous Disease of the Eye", by W. Camac Wilkinson, M.D., F.R.C.P.; 1933. London: J. and A. Churchill. Pp. 93, with 31 illustrations.

[2] "Science and Democracy: Adjusting the Laws of Advanced Mechanization to the Objectives of Civilized Policy", by Frank Trinca, M.B., B.S., 1933. Melbourne: Brown, Prior and Company, Limited. Royal 8vo., pp. 201.

The writer's thesis is that the main force operating upon modern industry and guiding its development is not finance, but science. Industry is aided, and in fact made possible, by scientific inventions. Entirely new discoveries are made at irregular intervals which completely revolutionize the industrial process and cause great bursts of prosperity in which productive output is magnified many times. But all production is to satisfy human wants, and "from any wave of invention there is a limitation in each age to the diversification of modes of living that it can confer on human activities". For this reason the machine temporarily reaches the limit of expansive work that it can perform and we are faced with "actual overproduction as the result of the machine overtaking demand". The sudden slump, as contrasted with the more or less gradual process of machine output reaching saturation point, he says, is caused by the operation of financial processes. All costs are inflated during the boom period as prices rise, and the faltering of production causes inflated values to fall headlong. In the recent crisis the questions of war debts, reparations, protective tariffs and similar factors all contributed to hasten the crash once the machine had been faced with its "biological limitations".

Human employment is grouped under three classifications: (i) the construction of capital assets; (ii) the production of perishable commodities, which are continually consumed; (iii) the conduct of administrative services. It is claimed that the slump occurs first in section one as each wave of invention reaches its crest, and spreads from there throughout the whole of economic society. The reaction is severe in industry of the third class, but, as certain goods are required at all times, production under class two does not suffer such an eclipse. The difficulty in industries producing consumption goods is that capital moves into this class out of class one, thus augmenting output at a time when the community's power to purchase goods is greatly depleted.

The writer then proceeds to discuss the financial question in detail. He briefly outlines the economic theory of "value" and "the quantity theory of money". But here his work lacks the sound reasoning which has characterized it to date. The attempt to deal with "value" in three pages is rather crude, while his treatment of the "quantity theory of money" is totally inadequate and constitutes a weakness in his thesis. Dr. Trinca's belief is that the quantity of credit or spending power is automatically expanded as production increases. This may be so, but the point which is overlooked is that quantity of money itself can generate a rise of prices, causing that stimulation of profits and appreciation of values which culminates in a boom followed by the inevitable depression. Finance is held to be inadequate in that it "handles commodities with its eye turned in the direction of distribution and its back to the machine process of production, which delivers its commodities in bursts and not in the uninterrupted cascade it is given credit for". It is thus, says Dr. Trinca, the duty of financiers to regulate credit so as to offset the wave-like motion of machine production, and to maintain gradual expansion as it now occurs between successive booms. The writer asserts that under no circumstances should the existing credit authorities be tampered with. Banking is a highly specialized and individual service, the operation of which requires wide experience. It should be left entirely in the hands of those who at present control it, as they are best fitted for the task. "The present system of finance may need, and is undergoing, great modifications in the present crisis. But the futility of any political debating junta assuming the rôle of modification can only be compared in its consequences to handing over the medical service to the faith healer." One cannot here resist the suggestion that medical practitioners who prescribe for economic ills may bear much the same relationship to the trained economist as the despised faith healer bears to the university graduate in medicine.

The next section of the book is particularly interesting to the student of economics, as it deals with that contentious subject, the part which should be played by government in the conduct of society. The writer allies himself with many of the leading economists, both past and present, in asserting that booms and depressions should be ironed out by means of an enlightened public works policy.

In the final section Dr. Trinca ventures to prophesy that the next great inventive wave will arise out of the understanding and full use of the atom. He sees cosmic energy so revolutionizing society that it will come to be regarded not as a democracy, but as a "cosmocracy". This dissertation is interesting, but might we not suggest that by the time physical scientists have succeeded in harnessing the atom, economic and financial scientists will so have improved the existing financial system as to remove the basis for criticism?

In the book there is much that is familiar to students of the trade cycle. Even the main thesis that scientific invention is the force generating booms is by no means new. Throughout we have the impression that the writer is concentrating too much upon this one factor, and is ignoring or brushing aside other important forces to which due consideration should be given in any work purporting to deal with the business cycle. When we consider the vast amount of careful research that has been done, an example of which is the work of Dr. Wesley Mitchell, who has published one book of 490 pages, who has almost completed a second volume, shortly to appear, and who is already giving thought to a third, we can only regard the present thesis as a drop in the ocean of knowledge on this fascinating subject.

BIOLOGY FOR CHILDREN.

"HOW YOU BEGAN: A CHILD'S INTRODUCTION TO BIOLOGY" is a clearly written little book by Annabel Williams-Ellis.[1] It gives an account in simple, humorous language of the development of the human body from the first cell to the complete infant, and shows how the baby at one stage in its development simulates many other forms of life.

Scientifically-minded parents will find it useful to read to their children, but the book is not true to its title, as it omits to explain the facts of birth and generation. When it is read to the average child, the mother will be bombarded with questions on these subjects. The small child, in whose language the book is written, is far more interested to know how he was born and why some women have babies and others do not than in the actual phases of the development of his body.

The subject matter of development and comparative biology appeals more to the adolescent, and the book would be more widely read if written for the older child.

THE STORY OF THE HUMAN BODY FOR CHILDREN.

"HOW YOU WORK", by Isabel Wilson, is a book that, once known, will become very popular.[2] It is a comprehensive work on anatomy and physiology written for children, and gives much valuable information in a most pleasing style. All boys and girls will easily be able to follow this book and enjoy it.

Dr. Wilson manages very cleverly to describe the structure and working of the body without using long names. She describes the various systems and the special senses, and also gives the child some idea as to how the body works as a whole in the last chapter.

This book should be included in the general reading of every boy and girl; and many adults, especially if they have been reared in a non-medical atmosphere, could read it with pleasure and profit.

[1] "How You Began: A Child's Introduction to Biology", by A. Williams-Ellis, with preface by J. B. S. Haldane; 1933. Australia: Angus and Robertson, Limited. Crown 8vo., pp. 91, with illustrations. Price: 2s. 6d. net.

[2] "How You Work: An Introduction to the Human Body", by I. Wilson, M.D.; 1933. Australia: Angus and Robertson, Limited. Crown 8vo., pp. 185, with illustrations. Price: 3s. 6d. net.

The Medical Journal of Australia

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THE PRE-SCHOOL CHILD.

To the intelligent observer the development and unfolding of the mind of a child are most fascinating. The learning of words and their correct use by a child who may have heard them only once, the remembering of incidents connected with these words, the use of the imagination and all that goes with it are a source of continual wonderment to those with the care of children. It is almost as interesting to watch the growth of the healthy child—the development of the sturdy little limbs and filling out of the body, both keeping pace with the tireless energy and *joie de vivre* that are typical of childhood. There is something lacking in the man or woman who cannot be moved by the joys and sorrows of childhood, by the restless longing of a child to see new things and to feel fresh thrills, by its pleasure in talking of them afterwards, and by its pathetic disappointment when wishes are thwarted and pleasures are not what it thought they would be. Many a woman and man who have said hard things about the coming of an unwanted child, have made a complete *volte-face* when it has arrived and begun to develop. But to be fascinated and interested is not necessarily to understand, and parents cannot do the best for their children unless they do understand.

The life of a child before puberty may be divided into four periods. There is the life of the *fœtus in utero*; then comes the period of infancy, of utter helplessness and dependence mainly on the mother; after this is what we may call the pre-school period, and finally there is the school period. Each of these has its peculiar problems and difficulties which have to be faced and dealt with by the understanding parent. At the same time each period has its medical problems and in each of them the parent has to rely for guidance on the medical practitioner. The medical aspect of three of these is recognized more or less by both parents and members of the medical profession. The need for ante-natal treatment has been preached for many years; ante-natal clinics are found at practically all obstetric hospitals and at some general hospitals; private patients, too, are coming in greater numbers to ask for supervision during the months of pregnancy. After the child is born, while it is helpless and dependent on the mother, medical aid is sought. Provision in this regard is made for certain patients at the public baby health centres, and many private patients voluntarily bring their babies to their regular medical attendants for advice on the question of feeding and so forth. During the school life of the child supervision is generally given, though in some places and in certain schools it is not adequate. The pre-school period alone, from the medical point of view, is often neglected. There is no organization, no branch of a health department, charged with the special care of the pre-school child, and parents seldom think it necessary to seek medical advice, chiefly because there is no obvious reason why they should. But the need is there. The pre-school period may be called the run-about period. It is the time when the child develops its immunity to infectious disease. It has to run the risk of infection from adults in the home; its tonsils probably become infected and perhaps

its adenoids as well, and a host of troubles may follow. During this period deficiency disease may become established and malnutrition may bring other sequelæ in its train. Teeth may become carious. Abnormalities in the eyes may be disregarded and defects in speech may make their appearance. Finally, problems of the emotional state and of behaviour may arise.

Many will say at once that most children survive this period without disaster. This may, of course, be true. Many homes are healthy and many parents are intelligent. On the other hand, many homes are not healthy and many parents are far from being intelligent; some apparently sensible parents become almost stupid where their children are concerned. Moreover, while some children do not meet with actual disaster, they are not so free from deficiencies, abnormalities and other troubles as they should be. The remedy lies in education. There is no organization that can deal with the matter, and it is doubtful whether an organization could be very useful. In America the National Congress of Parents and Teachers has held what they call a summer round-up of the children. Examinations have been made in a clinic, and medical practitioners have been helped in the examinations by nurses and voluntary workers. *The Journal of the American Medical Association*, in commenting on this work, stated that the group method had inherent defects that precluded the greatest benefits. With this we agree. The problems of each child are complicated and cannot be solved at a routine examination by a hurried and probably harassed medical practitioner. This is work for the general practitioner to do in the seclusion of his consulting room. But because people have to be taught that they should seek advice for their "run-about" children, progress will be slow. This is a subject that should be brought forward in health talks to the public; it might also be mentioned in talks given by education medical officers to such bodies as parents and citizens' associations. Medical practitioners in general practice can probably do as much as anyone else, if they will—they have many opportunities of teaching preventive medicine.

Current Comment.

HÆMOCHROMATOSIS.

HÆMOCHROMATOSIS is also known as "bronzed diabetes", but this term is not always applicable, as diabetes is not invariable and, when it does occur, is a late manifestation. Only about 150 cases have been recorded, the age period being from thirty to seventy years. Youth is spared and females generally escape. The lesions take some years to develop and clinical diagnosis may be impossible until the later stages. Cirrhosis of the liver is the first manifestation of the disease, followed by pigmentation of the skin and, later, sclerosis of the pancreas with diabetes. In the early stages the liver is enlarged, but later becomes smaller from contraction of the stroma. Ascites and jaundice may be present when the changes are rapid and there is blocking of the blood vessels and bile ducts. The spleen may or may not be enlarged. The pigmentation of the skin has been ascribed to hæmatogenous pigments, hæmosiderin and hæmofuscin, and secondarily to an increase of melanin from injury to the suprarenal glands by accumulation of hæmatogenous pigments. Primary liver cell carcinoma occurs in some cases, estimated at 10%. The primary cause of hæmochromatosis is not known. In some cases the liver has a high copper content. Cirrhosis of the liver has been observed in rabbits induced by copper poisoning, with lesions resembling those of hæmochromatosis and attended by deposits of hæmofuscin in the liver and other cells. This pigment gradually changes to hæmosiderin. E. S. Mills states that the records of his cases suggest, but do not prove, that the condition is due to copper poisoning from swallowing and inhaling copper dust, together with excessive alcoholic indulgence.

C. B. Rich[1] records a case of hæmochromatosis in which the heart exhibited pigmentation of the muscle cells and phagocytes in the interstitial tissue, with marked fibrosis, hæmorrhage and some fat infiltration. There had been pronounced heart failure with œdema, but it was not clear whether the failure was associated with the hæmochromatosis or was the aftermath of a previous rheumatic fever. There was no glycosuria, but the pancreas was not examined. Rich states that hæmosiderin contains iron, but that none can be detected in hæmofuscin. The relation of hæmoglobin of blood to these pigments, to the pigment of brown atrophy and to the yellowish brown material of advancing years is not definitely known. As most of the body iron is in the hæmoglobin of the red cells and as these are constantly being renewed, possibly there is increased destruction of these cells and faulty elimination of the iron. Pernicious anæmia is the only other disease in which hæmosiderin is deposited in quantity. But this disease shows nothing comparable to the marked degeneration of body cells in hæmochromatosis. In the latter the paren-

[1] *The Canadian Medical Association Journal*, July, 1933.

chymatous liver cells first accumulate the pigment; the Kupffer cells have none until enormous amounts fill the hepatic cells. The opposite occurs in pernicious anæmia. Destruction of old red cells and bile formation are not a function of the liver alone, but of the reticulo-endothelial system in general, of which the Kupffer cells are part. Probably the iron of hæmochromatosis is both from hæmoglobin and the body cells, which are the only sources of iron in the body. Rich does not accept the copper poisoning theory and states that in human hæmochromatosis there is no definite evidence of increased copper consumption and that investigators have failed to produce the disease in animals by giving large amounts of copper. Both copper and iron are needed to form hæmoglobin. Rosenthal suggests that the iron in the liver has a definite affinity for copper and that any increase in the liver copper is due only to the increase in iron; also that the liver cells are unable to reduce ferric to ferrous iron, which is the only form of iron usable by the body. Such inability, he states, is due to chronic infection of the colon injuring the cells and impairing their power of reducing iron. The unused iron, stored in the liver cells, accumulates and destroys the cells. Iron pigment is liberated and taken up by the resting histiocytes of the periportal area and by the Kupffer cells last. The degenerated hepatic parenchyma is replaced by fibrous tissue, forming periportal cirrhosis. The lymph glands join in the storage of the liberated iron, as do also the pancreas, spleen and myocardium. The deposition of hæmofuscin in the skin, muscle of the gall-bladder, intestine and heart Rosenthal explains as due to the action of the same toxin from the colon causing cell degeneration. Hæmosiderin is insoluble and is produced only by living cells in the presence of oxygen. If the body cell produces this substance it cannot excrete it and it will irritate and destroy the cell, the liberated hæmosiderin being caught by the lymphatic glands.

In pernicious anæmia the origin of hæmosiderin is from hæmoglobin destruction; in hæmochromatosis the body cell is suggested as its source. But Rosenthal assumes its origin from liver cells and not other body cells. Brown suggests its formation from the iron-containing protein of the liver cells independently of hæmoglobin. Ischiada considers that pigment may be formed in muscle from iron normally there. Rich suggests that there may be a general pigmentary degeneration of the body cells. Cirrhosis of the pancreas cannot be explained by irritation of the pigment present, as a similar deposit occurs in pernicious anæmia without cirrhosis. Rich suggests that the pigmentation and cirrhosis of the pancreas are due to the same toxin as affects the liver. Hæmosiderin is a product of degenerated cells. It is not seen in new cells and newly formed bile ducts. Fibrosis follows slow degeneration of the body cells. Hæmofuscin is also a product of cell degeneration. It has been ascribed to altered hæmoglobin or it may be related to the melanins. Rich considers it to be probably a product of the cell itself. The pigment of brown atrophy of the heart has been considered identical with hæmofuscin. It may be a melanin. In pernicious anæmia hæmosiderin comes from red cells and thus is deposited in the liver from an extraneous source. The poison inducing hæmochromatosis acts directly on the cells producing hæmosiderin and degenerative fibrosis. Rich remarks that the fact that the liver is involved primarily and constantly indicates the alimentary canal as the source of some toxin. It has been suggested that a chronic infection of the colon decreases the bactericidal and detoxifying powers of the alimentary mucosa, allowing bacteria or toxins to enter the portal circulation. But the liver normally contains more iron than other organs. In hæmochromatosis there is a deposit of an iron-containing radical and the liver would show the greatest change, no matter where the toxin originated, which might be any systemic source and not necessarily the alimentary canal.

Rich's speculations are interesting. He rightly refuses to accept copper as the cause of the condition. Our knowledge of the pharmacology of this metal does not warrant any such assumption. As regards the colon or other part of the alimentary canal, many sinister possibilities have been attributed to this organ, and to lay one more infamy to its discredit may not matter much. But we have no suggestion as to the identity of the mysterious organism producing the hypothetical toxin. Explaining the obscure by the more obscure is ever a profitless undertaking.

SUCCESSFUL REMOVAL OF A LUNG.

WHAT is claimed to be the first case of successful removal of an entire lung is reported by Evarts A. Graham and J. J. Singer.[1] The patient was a medical practitioner, aged forty-eight years, who was suffering from a squamous cell carcinoma of the bronchus of the upper lobe of the left lung. Before the diagnosis was made a pneumothorax developed as a result of an attempt to aspirate pus from the left pleural cavity. It was decided to remove the upper lobe of the left lung, but at operation the carcinoma was found to encroach on the bronchus of the lower lobe. For this and other reasons it was decided to remove the entire lung. Graham and Singer describe the steps of the operation. After removing the lung they removed seven ribs (the third to the ninth inclusive) from the transverse process of the spine to the anterior axillary line. After drainage of the cavity the patient left hospital apparently well; his only complaint was slight dyspnœa. The authors suggest that intratracheal anæsthesia was a factor of importance. Doubtless the fact that the lung was thrown out of action by the pneumothorax for some time before its removal had something to do with the result.

[1] *The Journal of the American Medical Association*, October 28, 1933.

Abstracts from Current Medical Literature.

MEDICINE.

The Systolic Murmur.

S. A. LEVINE (*The Journal of the American Medical Association*, August 5, 1933) deals with the significance of systolic heart murmurs. He classifies these murmurs in grades, from Grade I for the faintest definite murmur to Grade VI for a very loud murmur. He insists that a murmur should not be described unless it has an appreciable duration after the first heart sound. Among 1,000 patients examined at random a Grade III murmur was noted in 17 instances, and this was associated in every case with obvious cardio-vascular disease. In 196 patients a Grade I or II murmur was observed; in 45 of these no explanation could be found; in the remainder there existed obvious heart disease, a history of rheumatic fever, hypertension, anæmia, hyperthyreoidism or fever. In those patients in whom no obvious cause could be found the murmur was usually very faint, Grade I. The author tested ten normal men and found that after brisk exercise a systolic murmur of Grade I or II was noted; this murmur after exercise is apparently a normal phenomenon. Systolic murmurs either at the apex or the base of the heart are included in these investigations. He concludes that a systolic murmur of Grade I may not indicate disease, but that murmurs of Grade II or louder may be present with rapid heart action, anæmia, hyperthyreoidism, fever, mitral incompetence either due to organic disease of the valve or relative incompetence, hypertension, subacute bacterial endocarditis, nervous heart or aortic valve disease.

Chemotherapy of Cancer.

O. SCHÜRCH (*Deutsche Medizinische Wochenschrift*, September 29, 1933) writes of a series of 32 cases of carcinoma treated by him with iodide of lead (PbI_2). He records his results for two reasons: first, because iodide of lead has not before been used for this purpose, and secondly, because one of his cases shows how errors may arise in estimating the value of the results obtained by chemotherapy of cancer. The iodide of lead is insoluble in water, but soluble in calcium gluconate. For his injections he used 0·5% lead iodide in a 10% solution of gluconate. This preparation he designates "P.S.V." Animal experiments showed that "P.S.V." was much less toxic than any of fourteen other lead compounds that had been investigated by Bischoff, Maxwell, Evans and Nuzum. Of any of these ten to twelve milligrammes per kilogram of body weight was a lethal dose for a rabbit, whereas of "P.S.V." twenty to thirty milligrammes per kilogram was well borne, though it produced a fall in hæmoglobin and body weight. These returned to normal in four or five weeks. In treating his patients the author used gradually increasing intravenous injections of "P.S.V.", the maximum single dose being ten cubic centimetres of the solution, and the total amount in the course one hundred cubic centimetres, that is, 0·5 gramme of lead iodide. The series comprised: cancer of stomach twelve cases, of sigmoid and rectum seven, of breast five, of larynx two, of tongue two, "*struma maligna*" three, pancreatic tumour one. These were all inoperable cases or were characterized by recurrences or metastases. The author's brief summary of the results of treatment is that in no single case was any objective improvement demonstrated. Sometimes there was a temporary subjective improvement. But even in these cases the disease advanced without any perceptible retardation. The toxic effects of the lead comprise reduction of red cells and of hæmoglobin, the presence of pathologic red cells, and loss of weight. In general, "P.S.V." was well tolerated. Such symptoms as followed an injection were probably attributable to the calcium rather than to the lead. And it is to the calcium that the author attributes the temporary subjective improvement that many patients experienced. He considers that any good effect obtained by lead therapy of cancer is dependent on the toxicity of the lead preparation used and on the systemic damage thereby produced. In comparison with other lead preparations his "P.S.V." had a low toxicity and its cancer effects were correspondingly slight. One patient remained well three years after operation and subsequent lead treatment. This was a woman aged fifty, with a cystic tumour of the tail of the pancreas. At operation this cyst was easily shelled out and was considered to be non-malignant. On subsequent pathological examination it was reported to be an "adenomatous carcinoma". Although a pancreatic fistula persisted for four months after operation, there was no recurrence of the growth.

Skin Temperature.

J. JPSEN (*Münchener Medizinische Wochenschrift*, June 23, 1933) used the following technique for measuring skin temperature. A felt cloth, five by five centimetres and one centimetre thick, is fixed over the part to be measured and a thermometer (scale 24° to 42° C.) is placed between the felt and the skin for fifteen minutes before being read. The felt should be fixed to the skin for a quarter of an hour before measurement. It is important to see that the mercury container of the thermometer is completely covered by the felt. If a person is confined to bed with a bad knee, the felt is fixed to the knee and the temperature is measured every three hours (five times) and the average is taken. If the temperature varies by 1° C. in the middle of the day, it suggests an abnormal state. To be accurate, the temperature should be measured on four days and the average taken. The author's conclusions are as follows: (i) In a case of embolism the skin of the affected leg registered 27·4° C. as compared with 36·2° C. on the healthy leg one and a quarter hours after its occurrence. He regards this finding as significant. (ii) In a series of cases of diabetic or of infectious gangrene the diseased leg was warmer than the healthy one. From this one can conclude that the gangrene is not due to a diminished flow of blood; conservative treatment can here be used. (iii) Arteriospasms can be diagnosed occasionally. They occur frequently and are one-sided. If the skin temperature sinks below 30° C. the person has cold feet, and if below 26° C. he complains of pains. (iv) In doubtful joint affections one can, by measurement of the skin temperature, decide whether there is a pathological change in the joint. Measurement of skin temperature is also useful in a doubtful tuberculous joint. (v) In phlebitis the skin temperature on the affected side is elevated. One finds regularly in pregnant woman, also in the puerperium, a maximal elevation of the temperature of both extremities, and therefore one cannot expect a further elevation of temperature if phlebitis occurs. (vi) In thyreoid disease the foot temperature rises and falls, varying with metabolism. (vii) In anæsthesia one finds on loss of consciousness a marked rise of the temperature of the feet. This sign, in the author's opinion, is the surest sign that the patient is asleep. If the temperature does not rise or if a second fall occurs, this means that a condition of shock is present or threatening and treatment is necessary.

Sciatic Irritation.

P. C. WILLIAMS (*The Journal of the American Medical Association*, November 12, 1932) discusses reduced lumbo-sacral joint space in relation to sciatic irritation. One hundred and seven patients with sciatica were examined; lateral and antero-posterior X ray pictures were taken in all cases. A narrowing or a complete loss of the intervertebral disk between the fifth lumbar and first sacral vertebræ was noted in the majority of patients (59). The patients were chiefly young adults, the symptoms commonly appearing about the age of thirty. The fifth lumbar and first sacral nerve segments are intimately related to the lumbo-sacral junction. The fifth lumbar nerve may be compressed in the intervertebral foramen, owing to the slight deformity resulting from the narrowing of the lumbo-sacral joint. The pain complained of was low down in the back, along the postero-lateral aspect of the thigh and calf and on the dorso-lateral aspect of the ankle and foot. Other bone disorders associated with sciatic and low back pain were sacro-iliac arthritis, spondylolisthesis, lumbo-sacral anomaly, and hypertrophic arthritis (15 cases). The last mentioned condition was noted in later life. Treatment consisted in attempt-

ing to limit movement of the affected joint by plaster jackets or canvas corsets; if the patient was unrelieved, a plaster spica was applied from the axillæ to the toes and to the knee on the unaffected side. Relief was obtained in forty-eight hours. After two or three weeks the spica was removed, a canvas corset was fitted, and massage and baking were given to the lower part of the back. In resistant cases immobilization was obtained by operation to produce lumbo-sacral fusion.

Phrenic Paralysis by Injection of Alcohol in Pulmonary Tuberculosis.

L. CORDEY AND P. PHILARDEAN (*Revue de la Tuberculose*, June, 1933) analyse the results obtained in sixteen patients suffering from pulmonary tuberculosis, whom they subjected to temporary hemidiaphragmatic paralysis by injecting alcohol into the phrenic nerve. In only one patient had the diaphragm recovered its function within six months. In three others normal function returned within one year. Paralysis had persisted in two patients at the end of two years, and in two at the end of three years. If there was doubt as to recovery, electrical stimulation was employed as a final test. The authors emphasize certain points in technique, such as exposing a sufficient length of nerve, avoidance of stretching of the nerve, the use of absolute alcohol, and injection of the nerve in several places. With injection of five centimetres of the nerve, employing absolute alcohol, they postulate paralysis of at least twelve months' duration. They admit that paralysis may rarely be permanent, and they also recognize that a secondary phrenic avulsion is exceedingly difficult, as the nerve is buried in scar tissue. In only one patient was there secondary ill effect, namely, brachial pain for some three weeks. The authors conclude that this operation has many advantages over phrenic avulsion, in that it accomplishes as much as the latter in healing a pulmonary lesion, while at the same time permitting a restoration of diaphragmatic function. They decide, therefore, that it is deserving of more frequent employment.

Immunity in Malaria.

H. STOTT (*The Indian Medical Gazette*, September, 1933) discusses the mechanism of immunity in malaria. He points out that immunity in protozoal diseases, such as malaria and kala-azar, differs greatly from immunity in bacterial diseases, such as typhoid fever, in that comparable toxin production and the development of antibodies are not known to occur. The enlargement of spleen and liver suggest that the reticulo-endothelial system may be concerned in the production of immunity in both malaria and kala-azar. Phagocytosis of malarial pigment can be readily demonstrated in smears from the bone marrow, liver and spleen of a person who has died of malaria. In malaria of birds and monkeys phagocytosis of plasmodium has been shown to occur in liver and spleen, and also, if the infection is heavy, in the peripheral blood. Before December, 1932, however, there had been very few recorded cases in which malaria parasites undergoing phagocytosis had been observed in the peripheral blood of man. The author remarks that he has seen the phenomenon on two occasions, once in 1919 and again in 1932. Rings of *Plasmodium falciparum* and remnants of sporulating bodies were observed in the cytoplasm of large mononuclear cells. In every case of which there are clinical records the infection was a very heavy one of malignant tertian malaria, and there were sporulating bodies in the peripheral circulation. The author suggests that phagocytosis normally takes place "in the fixed reticulo-endothelial cells", and that these cells become free in the blood only when the reticulo-endothelial tissues are excessively stimulated by very severe infection.

Monkey Malaria Treated by "Atebrin".

R. N. CHOPRA AND B. M. DAS GUPTA (*The Indian Medical Gazette*, September, 1933) discuss plasmodium infection of monkeys and its treatment by intravenous and intramuscular injections of "Atebrin". Napier and Campbell found in the blood of a Malayan monkey (*Macacus irus*) a parasite resembling the parasite of human malaria. This monkey's health did not appear to be disturbed by the infection; but *Macacus mulattus*, when inoculated intravenously with the plasmodium, became acutely ill and soon died. *Semnopithecus entellus*, the common "hanuman" monkey of India, is also highly susceptible; *Macacus radiatus* and *Hylobates hoolock* are only slightly susceptible. Knowles and Das Gupta succeeded in transmitting the infection from monkey to man. The authors remark that the plasmodium, which has been named *Plasmodium knowlsi*, is more closely related to human plasmodia than the various plasmodia of birds; monkeys infected with it should therefore be more suitable for testing the action of anti-malarial drugs. The authors inoculated twelve monkeys of the species *Macacus mulattus* with *Plasmodium knowlsi* and treated them for their resulting illness by intravenous or intramuscular injections of "Atebrin". The weight of the animals varied from 3·2 to 4·7 kilograms, excepting in two instances, in one of which the weight was less than two and in the other greater than five kilograms. The average daily dose of "Atebrin" was 0·025 gramme; larger doses produced ill effects; doses of 0·04 to 0·05 gramme given intravenously invariably caused death in ten to twenty minutes. Although treatment was not administered until the infection had become very heavy, two or three doses were all that were required to cause the disappearance of the parasites from the peripheral blood. Schizonts and gametocytes were alike affected. The authors noted evidence of degeneration of the parasites after only one injection of "Atebrin". Treatment with "Atebrin" did not prevent relapse. The parasites reappeared in the peripheral blood ten to fifteen days after the cessation of intensive treatment, such as the administration of large doses of the drug over a period of five days. In the relapses the parasites multiplied with the same rapidity as in the primary attack and caused death if prompt treatment was not instituted; but relapses were more readily controlled than the primary attack.

Agranulocytic Angina.

F. L. FORAN, H. M. SHEAFF AND R. W. TRIMMER (*The Journal of the American Medical Association*, June 17, 1933) discuss the treatment of agranulocytic angina. There is a widely current view that this condition is characterized by defective function of bone marrow, hence there is a logical basis for treatment with liver extract. Preparations of the fraction G of Cohnheim were injected intravenously in the acute cases, intramuscularly or orally in the less acute. The equivalent of 100 grammes of liver was injected every eight to twelve hours until a definite rise occurred in the total white cells or granulocytes, or until definite clinical improvement occurred. In five patients treated the equivalent of 2,000 grammes of liver was injected. In each patient ulceration about the mouth or throat was noted, associated with a white cell count of 2,500 per cubic millimetre or less. Granulocytes or polymorphonuclear leucocytes were reduced in every patient to 17% or less. All these patients recovered and remained well, except one, who died in a recurrence of granulopenia during an attack of lobar pneumonia. In all patients except this one the leucocytes and granulocytes increased after four to eight days in a similar way to the increase of reticulocytes in pernicious anæmia under the same treatment.

Hereditary and Constitutional Factors in Leuchæmia.

P. MORAWITZ (*Münchener Medizinische Wochenschrift*, August 4, 1933) reports two cases of chronic lymphatic leuchæmia occurring in two brothers aged fifty-seven and fifty-five years respectively. Both the localization of the disease and the blood findings were similar. This observation, as well as records in the literature, make it probable that the author's findings are not merely a coincidence, but rather due to an hereditary constitutional factor. Chronic irritation is accepted as the probable cause of leuchæmia in radiologists. Another interesting fact is that the types of leuchæmias in the family may be different, and this also applies to leuchæmia occurring in radiologists, although the ætiological factor is the same.

Special Articles on Treatment.

(Contributed by request.)

XXIII.

VARICOSE ULCERS.

NOTHING is more worth while than the relief of the common ailments of life. The brilliant surgeon who can remove a ruptured ectopic in a few hectic minutes and by injecting the shed blood into the veins restore life to a moribund patient, must have a special niche in the hierarchy of the great; but the general practitioner who is successful in the relatively sordid task of curing chronic ulcers of the leg confers such a boon on such a number of plodding sufferers that, in the aggregate, his work will entitle him to equal honour.

I had the good fortune to be in London in 1929 when Dickson Wright was perfecting his technique in the treatment of varicose ulcers, the details of which are so lucidly set out in *The Lancet* of February 28, 1931. Always having had a hobby for "bad legs", I was able to appreciate and make good use of his principles and turn this hobby into a very profitable specialty. My aim in this paper is not to recapitulate these principles, but to emphasize their fundamental importance and to pick out certain difficulties and variations that arise in their application.

Regarding the ulcer itself, the term "gravitational" is probably the most comprehensive, and the realization of the asphyxial element the most useful mental approach.

A knowledge of the physiology of the capillary circulation, particularly in its relation to the tissue spaces and fluids and the lymphatic reticulation, is essential to a proper understanding of the lesion. The actual chemistry involved is not known and is unimportant from the standpoint of treatment. As to the anatomy involved, the general knowledge is sufficient, emphasizing as it does the importance of the communicating veins between the superficial and the deep vessels, the rôle of the valves, and the supporting function of the subcutaneous tissue and the skin.

Where the stasis leading to asphyxia is due to varicose veins, the aim is to remove the back pressure by obliterating the offending vessels. Where cardiac dropsy or other circulatory factors are the cause, the treatment must be directed along constitutional lines in addition to the local effort. There is a type of ulcer-bearing leg frequently found in thin elderly women that has neither varicose veins nor œdema and constitutes the most difficult of all to treat. Diseases such as syphilis, while complicating the issues, are not in general to be regarded as the cause of these ulcers.

Whatever the underlying cause of its chronicity may be, the actual ulcer is always traumatic in origin. So long as the skin is intact the symptoms are limited to a dull ache and a sense of weight and, especially if œdema be present, to the irritation of an attendant eczema; but the least break in the continuity of the surface forms a focus from which the process continues to spread until the escaping fluid allows an approximate equilibrium to be maintained. Usually an infection of varying intensity quickly supervenes.

In broad terms, the basis of Dr. Wright's treatment is to apply at once, from the toes to the knee, adhesive, impervious, more or less elastic bandages (best represented by the "Elastoplast" brand), at the same time obliterating by injection any varicose veins that may be present. In these circumstances infection is adequately dealt with by the normal body processes.

The degree of infection frequently amounts to a definite cellulitis about a foul sloughing mass. In such a case it is not common sense to follow Dr. Wright blindly and to apply at once an "Elastoplast" bandage, nor would one court disaster by injecting a vein in such a field. It is equally unwise to follow his advice in the œdematous leg and apply a bandage "with all one's strength". It is surely asking very little to put these patients to bed for the few days required to combat the active inflammation or œdema before commencing the actual treatment. I have further found the application of powdered aspirin before bandaging, as advocated by him, to cause intense pain and a nasty, rebellious eschar. An ulcer which has, however, been fairly well looked after by the patient may be covered at once, and if the œdema is only moderate the use of an "Elastoplast" bandage applied in the early hours of the day is permissible; but in no case should one's enthusiasm be allowed to violate the established canons of rational therapy.

The intervals at which the bandage requires to be changed vary with the individual and the type of the ulcer. Sometimes the discharge from the ulcer seems to be particularly irritating to the surrounding skin, and frequent changes (at least weekly) are desirable. This fits in with the appropriate period for injecting related veins and is probably the most generally convenient. Some skins become eczematous under the bandage, while others develop an accumulation of pasty sebaceous material exactly like the *vernix caseosa* covering certain new-born babies.

Always before applying and immediately after removal of the "Elastoplast" a liberal swabbing with methylated spirits should be given. This is not only cleansing at the time, but helps to keep the skin healthy. Where irritative pitting develops, the sparing application of bismuth formic acid powder is very useful. Some cases of sensitive skin appear to do better with lint or other material under the bandage. Needless to say, the greatest importance attaches to the even application of the bandage and judgement of the correct pressure to be used. Even this requires practice, and it is better to acquire the necessary skill oneself than to leave it to a nurse. It should never be relegated to the patient.

If there are a number of veins to be injected, skin grafting need not be considered, as the ulcer will be healed before the series is completed, but in certain cases of large ulcers with few veins the most dramatic results may be obtained. The following case has a double lesson.

Mrs. J.W., aged fifty-four years, had had for many years an extensive ulcer on each leg, at least ten centimetres (four inches) across and almost encircling the limb. When I saw her the veins had been well injected by a colleague, but to the patient's disappointment the ulcers were as bad as ever. They were really bad, painful to a degree, and foul smelling. After three days' fomenting with potassium permanganate I applied "Elastoplast" bandages and, as the pain was intolerable, prescribed a sedative mixture. With the greatest difficulty the patient was persuaded to wear the bandages for a week. On their removal a clean granulated surface presented. A Reverdin graft was done immediately, points of skin from the thigh being taken under local anæsthesia and fresh bandages applied. At the end of another week, on removing the bandages every graft had taken well and the discharge was much less. A third set of bandages was applied, and on their removal a week later the grafts had coalesced and the ulcers were healed. The legs were then left lightly bandaged for another two weeks to allow consolidation to take place, after which, with the exception of some massage, no further treatment has been required.

Regarding the injection of the associated varicose veins, this has been so thoroughly dealt with in a recent number of the journal (September 30, 1933) that little need be said. After thoroughly trying all the preparations commonly advocated I have found quinine-urethane the most generally useful, with sodium morrhuate for those cases in which the former is contraindicated. When a large number of injections are being given it is most convenient and cheaper to have the solution prepared according to Martindale's formula in considerable quantities. In the winter there is the slight disadvantage of having to heat up the solution before using it, but this is compensated by the constancy of the results. Three cubic centimetres may be injected into the larger veins with impunity if a proper technique is followed. If one injects a vein filled with blood the consequent dilution of the solution will obviously affect the result. Equally obviously, if one elevates the leg after injection some of the solution will reach the general circulation and con-

stitutional symptoms must be expected to occur. If, however, the patient be seated on the couch with the legs outstretched at right angles from the body, there will be sufficient pressure head in the veins to prevent a centripetal flow of the solution, and not too much to prevent the vessels from being virtually emptied by manipulation before injection. The use of a constricting band is quite unnecessary. The injected fluid, being directed distally and helped in its distribution, if necessary, by manipulation, is largely fixed by its reaction on the endothelium, and when the patient resumes the upright position it is, from the very nature of the case, confined to the vein injected. Excessive periphlebitis following injection must make one suspect one's asepsis, but a successful reaction must be more or less painful, especially at the commencement of the day's work. Bandaging and the application of roller pads over the vein do very little towards diminishing the size of the resultant mass. A large vein will give a large mass whatever procedure be adopted.

One must not look for perfect consistency in the reaction obtained. At times an injection is quite unsuccessful, but on being repeated a week later an ideal result may follow. The large bunches of veins frequently found in the calf of the leg are the most disappointing, chiefly owing to their communicating branches and consequent loss of solution. The small veins about the ankle are difficult, often appearing to have a definite power to avoid the needle. These are not, however, true varicose veins, and with the relief of the back pressure by the injection higher up in the limb will in general cause no symptoms. The fine spider web venules, wherever situated, can be successfully relieved only if their feeder vein can be sealed. Prominent thin-walled veins with atrophy of the overlying skin should always be approached from an angle. Even so, their covering will often break down and a dark semi-fluid discharge occur. This should be evacuated as completely as possible, when healing will follow. Should leakage of the solution into the subcutaneous tissue occur during injection, allowing bleeding for a few minutes will sometimes clear it out. With quinine-urethane the consequent sloughing is very intractable, but fortunately painless. Fortunately, too, most patients take such a happening philosophically. With experience such accidents become very rare. The best way to avoid them is to use a Jones's syringe, which immediately registers the entry to the vein. The patient's sensations during the actual injection are a valuable guide, any pain beyond that of the initial puncture being a danger signal. After the injection the vein may be compressed between two fingers spread like a "V", with the puncture between them, and a drop of collodion applied directly to the skin. The patient should remain in position for a few minutes.

Results are in general so good, despite the rather haphazard methods followed, that many obvious questions remain unanswered. Chief among these is that of the period that should elapse before the full volume of blood is allowed to flow back into the leg by the resumption of the erect attitude. When a definite spasm of the vein is seen immediately after injection one may count on a good result, and I always like to wait for this before allowing the patient to stand up.

Concerning after-treatment, in the simple case with the veins successfully sealed and the ulcer healed, practically no after-treatment is necessary. Swabbing the skin daily with methylated spirits for a time restores the tone and a *crêpe* bandage may be advised as a protection until confidence is restored. Where eczema has been a prominent symptom, treatment for this may have to be continued, but with the prognosis of a complete cure. Where œdema of a constitutional type is present, an elastic support may have to be worn more or less permanently. If recurrence is to be avoided in these cases the leg must simply not be allowed to swell. I supply a three-inch elastic web bandage, which the patient must adjust before rising in the morning, and if the case be a bad one, I insist on a quarter of an hour's elevation of the leg every hour of the day. This is at first irksome, but the patient soon learns that more work can be done in the remaining three-quarters of an hour, and without discomfort, than by any other method. Properly performed massage is of great value in the after-treatment of certain cases.

Conclusion.

The primary consideration in the treatment of gravitational ulcer is to remove the cause. In generalized œdema this is best done by the application of "Elastoplast" bandages, which have the double function of supporting the back pressure in the limb and temporarily replacing the missing skin, thus restoring the static equilibrium in the tissue spaces and allowing natural repair to take place. After-treatment, both local and general, is necessary to prevent recurrence. In true varicose ulcers the essential element is the obliteration of the offending veins, the application of the bandages being as much as anything to keep the patient and his many ointments away from the field. Any good adhesive strapping will suffice, but the "Elastoplast" is of outstanding usefulness. After-treatment is aimed at improving the health of the damaged area. Skin grafting is useful in large ulcers with few veins.

A. E. PANTING, M.B., B.S., B.Sc. (Melb.),
Honorary Surgeon to Out-Patients, Honorary Surgeon to Special Clinic for Diseases of Skin and Veins, Launceston Public Hospital, Tasmania.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Warrnambool on July 22, 1933.

Rest and Movement.

DR. FAY MACLURE read a paper entitled "Rest and Movement". This paper, with the discussion that followed its reading, will be published in a subsequent issue.

Toxic Thyreoid Adenoma.

DR. H. I. HOLMES showed a woman, aged forty years, who gave a history of having developed enlargement of the thyreoid gland in 1912 after a severe mental shock. In 1913 she complained of weakness and palpitation. The pulse rate was 124 in the minute. The thyreoid gland was somewhat enlarged; there was no definite exophthalmos. As the pulse rate was persistently over 120, "Thyroidectin" was given and the rate fell to 108. In 1914 the patient's appendix was removed. In 1915 the patient submitted to an Alexander-Adams operation; the thyreoid gland was still somewhat enlarged. In 1918 the patient complained of gastric symptoms. An X ray examination was made and the diagnosis was "adhesions to the large intestine and gastroptosis".

On May 20, 1926, it was noted that the right side of the thyreoid gland became enlarged and that the enlargement subsided. The patient felt tired after "attacks". The pulse rate, which was 160, fell to 120 while the patient was resting. A small adenoma was found in the right lobe of the thyreoid and a diagnosis of toxic adenoma was made. The systolic blood pressure was 170 and the diastolic pressure 120 millimetres of mercury. On September 16, 1926, the right half of the thyreoid gland, together with the adenoma, was removed. The pulse rate did not become slower. The patient's condition improved later, and while she was taking calcium lactate and "Thyroidectin" the pulse rate fell to 100. After this the patient's condition varied. Enlargement of the left lobe became evident and the enlargement varied from time to time. At intervals a mottled rash appeared on the chest with dermographia.

The patient was in hospital for one month in March, 1932, and some general improvement resulted. A recurrence of symptoms that occurred later was relieved with "Luminal", rest *et cetera*.

On March 10, 1933, the patient's pulse rate was 160, and immediate relief was obtained with "Amytal". The patient was in hospital for two months and was treated with ephedrine and later with "Tricalcine" and Lugol's solution. Dr. Holmes asked whether the symptoms were attributable to the thyreoid and, if so, whether operation was likely to be followed by relief. Dr. Holmes said that he was in doubt as to how many of the symptoms were psychic and how many were due to thyreoid intoxication.

DR. S. O. COWEN said that the case was a most interesting one and demanded more careful study than he had been able to give it in the time available. He was inclined to Dr. Holmes's view that the greater part of the patient's present illness was "functional" or "psychic" in origin. The failure to improve after the removal of the adenoma in the right lobe of the thyreoid, her long history, and the failure to respond to specific thyreoid treatment, and finally the rapid improvement on "Luminal" and other sedatives were the factors on which Dr. Cowen based this opinion. Nevertheless, he thought that, if only for its value as suggestion, the adenoma in the left lobe of the thyreoid should be removed and that the operation should be preceded and followed by appropriate psychotherapy.

DR. LESLIE HURLEY said that it was not possible to express a definite opinion after only one examination in such a difficult case, but there were several points in evidence against the patient's condition being one of neurasthenia as against thyreotoxicosis. The patient slept with two blankets only, even on cold nights, and on examination her skin was fairly warm in spite of the cold day. The thyreoid gland enlargement had increased during the last twelve months and the patient had a bright appearance suggestive of thyreoid toxæmia rather than of neurasthenia. He recommended an estimation of the basal metabolic rate and thought that if the rate were raised operation would be advisable. He considered that to perform a further operation on the thyreoid gland as part of psychotherapy was a drastic and unnecessary proceeding.

DR. N. A. ALBISTON said that he thought there was no question but that some of the patient's symptoms were due to excess of thyreoid activity of some sort. The question whether the cause of this excessive activity was chiefly physical or psychological was not vital in the present case. It had been said that physical and mental influences met in the endocrine glands. The condition of anxiety neurosis, as in the present case, might produce a psychogenic thyreotoxicosis that would present a clinical picture identical with the picture produced by physical causes. In the present case Dr. Albiston considered that both physical and psychotherapeutic measures should be employed and he thought that a thorough psychological investigation was indicated, but psychotherapy unaided by direct treatment of physical symptoms would probably fail.

DR. H. F. MAUDSLEY agreed with Dr. Albiston. Neurasthenia and anxiety neurosis were not identical. He would prefer to make a more certain diagnosis of causation before advising operation, and thought it would be interesting to see the result of a course of treatment with Lugol's solution.

DR. M. D. SILBERBERG considered that the case came within the psychotoxic group. "Snappy" heart sounds were typical of these cases. The patient's raised blood pressure and slight enlargement of the heart were also consistent with thyreoid intoxication. He considered that the psychological background should be explored before further operation was undertaken, and in his experience many patients of this type improved on Lugol's solution combined with "Luminal".

DR. J. HAYDEN agreed with Dr. Silberberg that there was a definite thyreotoxic element in the case. He considered that the patient should be operated on.

DR. GEOFFREY PENINGTON said that after the mental shock of several operations the presence of a lump in the left lobe of the thyreoid was sufficient to precipitate further anxiety and so thyreotoxicosis. He thought that it would be impossible to restore the patient to normal health without further operation, but before doing this he thought it would be essential to have an estimation of the basal metabolic rate.

DR. W. A. HAILES said that after a rather brief physical examination of the patient he had formed the opinion that the tumour in the left side of the neck was a nodular goitre giving rise to thyreotoxic symptoms. The palpable tumour was probably an area of hyperinvolution and this could best be dealt with by performing a subtotal lobectomy. In his experience the psychic factor in causation was more common in those patients presenting a more uniform thyreoid enlargement rather than the present type with nodular enlargement.

DR. A. E. COATES agreed with Dr. Hailes that the condition was a nodular goitre and that the operation of subtotal lobectomy should be performed.

DR. VICTOR HURLEY said that he thought there was enough evidence to diagnose the condition as a thyreotoxic goitre. The failure of the patient to improve after removal of the right lobe was not proof of the absence of this condition. He himself had been forced to remove the second lobe in three somewhat similar cases, ten, fifteen and seventeen years respectively after the first lobe had been removed by Sir Thomas (then Dr.) Dunhill. These patients had complained of recurrence of the symptoms of thyreotoxic excess and in one case of pruritus. The history of severe mental shock was frequently a feature of the onset of thyreotoxic symptoms. Dr. Hurley said that he would not expect a dramatic improvement after a further lobectomy in the present case, as in such patients the period of improvement had some relation to the period of preceding illness.

DR. F. L. DAVIES said that, speaking not only as an anæsthetist but as an observer of thyreoid surgery for a large number of years, he had a great personal interest in the subject under review. Even before the Australasian Medical Congress in 1923 certain Sydney surgeons had pointed out that it was necessary to remove more than half the goitre in order to achieve permanent therapeutic results, and the present patient seemed to illustrate this contention. With regard to the anæsthetic risk in a patient of this type, gas anæsthesia was probably a little safer, but consideration had to be given to local conditions, and in his opinion the operation could be performed safely under local anæsthesia, gas anæsthesia, or ether given by the open method, and the choice would be with the surgeon, having regard to his available anæsthetist. Dr. Davies would not look upon this patient as presenting a grave anæsthetic risk.

In concluding the discussion, Dr. Holmes said that at the time of the first operation there had been no obvious pathological condition of the left lobe.

Carbuncle of the Kidney.

Dr. Holmes also showed a man, aged thirty-nine years, who, about three months ago, developed a sore on the back of the right hand with pin-head pus and increasing in size. Then, for about another month, he was troubled with "carbuncles" on the arms; he had eight altogether. No surgical interference was undertaken. The patient's temperature was 38·3° C. (101° F.). Later he complained of pain in the left lumbar region and general malaise and fever. He was in bed for two weeks prior to admission to Warrnambool Hospital on June 16, 1933. He was under observation for a fortnight, and carbuncle of the kidney was suspected. No urinary symptoms were present; there was no pus in the urine. Cystoscopy and the dye test showed normal ureteral openings and excretion of dye in normal time, the left excretion being less intense. X ray examination showed an indefinite outline of the left kidney. The patient had an irregular temperature that persisted; the pulse rate was about 70 to 80. Some pain at times and tenderness were present in the left loin, but were not pronounced. A needle was inserted, but no pus was found.

Operation was performed on June 29; left nephrectomy was performed. A large carbuncle was found on the convexity of the kidney. No accumulation of pus was present and a fatty capsule was adherent over the kidney. A drainage tube was inserted. A culture of pure *Staphylococcus aureus* was obtained.

The patient had an irregular temperature for two weeks after the operation; some infection of the wound occurred and during the third week the temperature steadily rose and reached 40·8° C. (105·4° F.) (taken in the rectum), and the patient looked very ill. Signs suggesting pneumonia were present at the right base. Anti-pneumococcic serum was given. X ray examination of the chest failed to reveal pneumonia, but suggested medi-

astinal trouble. Three needles inserted into the right side of the chest near the base yielded no pus, but slightly blood-stained pleural fluid. Next day 10 cubic centimetres of antistaphylococcal serum were given. The temperature reached normal in two days, with rapid improvement in the general condition. A cardiac bruit was heard.[1]

In commenting on the patient's present condition, Dr. Holmes said that the improvement following operation had not been as good as he had hoped, but following the administration of one ten-cubic centimetre dose of antistaphylococcal serum (Commonwealth Serum Laboratories) hypodermically the improvement had been definite, though it was not possible to say whether the improvement was really caused by the serum.

DR. HENRY N. MORTENSON said that the specimen of carbuncle of the kidney was a very good one. Dr. Holmes had been fortunate in seeing four patients suffering from this condition during the last few years. A study of the literature would suggest that it was a comparatively rare condition. The history following operation in the present case was not what one would have expected. He thought that the improvement following the administration of serum was temporary only, and he did not feel confident that this improvement would continue. He was afraid that a relapse would occur and that the outcome would be unfavourable.

DR. W. A. HAILES said that he had seen only one patient suffering from carbuncle of the kidney before, and this patient recovered after operation for its removal. In the present case he thought that the improvement had been due to the administration of serum and that the patient was now going to get well. He intended to try the more extensive use of antistaphylococcal serum in his own patients, particularly in the septic wards at the Melbourne Hospital.

DR. M. D. SILBERBERG said that at the present time at the Alfred Hospital there was a boy suffering from meningitis. His cerebro-spinal fluid contained leucocytes, albumin and *Staphylococcus aureus* and influenza bacilli. He had been given antistaphylococcal serum intrathecally and later intramuscularly. At the present time the patient bade fair to recover, though the intrathecal injection had been discontinued after it was found that the serum contained tricresol.

DR. THOMAS KING asked if it was antibacterial or antitoxic serum that was used, in reply to which Dr. Holmes produced a package of the serum, from which it appeared that it was antibacterial.[2]

DR. LESLIE HURLEY said that an important question at the present time was what had been the cause of the post-operative rise in temperature. Was it due to a localized infection or a blood infection? He hesitated to attribute the dramatic result to such a small dose of serum administered after five weeks of illness. There was no doubt about the presence of free fluid in the patient's peritoneal cavity, and this might be the cause of the rise in temperature. He felt that the present condition might be either a peritoneal infection or a heart valve infection.

DR. A. E. COATES congratulated Dr. Holmes on his diagnosis of the case. Regarding treatment, there was evidence that septicæmia was still present. The patient still looked ill and might at any time develop a metastatic infection, such as arthritis or empyema. One patient he had seen seemed to have been helped by antistaphylococcal serum in doses of ninety cubic centimetres given every second day.

DR. G. PENINGTON said that up to date experimental and clinical evidence went to show that staphylococcal antibacterial serum was of no value. The damage in staphylococcal infections was due chiefly to the toxins elaborated by the organisms, and in recent British journals it was stated that true antitoxic serum was of definite value.

Exophthalmic Goitre.

Dr. Holmes also showed a woman whose history began in 1929, when, at the age of twenty-seven years, after two weeks' treatment with Lugol's solution, she had the right lobe of the thyreoid removed on August 23, 1929. A fibrous capsule made removal more difficult than usual.

On April 22, 1930 (eight months later) the left lobe was removed, the isthmus and a fragment of the lobe being left. The remaining portion of the thyreoid isthmus became much enlarged and was pressing on the trachea; the pulse rate was 130. On June 19, 1931, the remaining part of the thyreoid was removed with difficulty.

On March 12, 1932, a small tumour situated to the right and below the hyoid bone was discovered. It was thought that this was either an accessory thyreoid or was the result of hypertrophy of a small remnant at the upper pole of the right lobe. On March 10, 1933, this small tumour was much larger and harder, and was about the size of a walnut. On July 5, 1933, the tumour had diminished in size to about half. The patient's general condition was much improved and she could follow her occupation, though she was inclined to dyspnœa on exertion.

Intestinal Obstruction Caused by Meckel's Diverticulum.

DR. A. E. BRAUER also showed a male patient, aged thirty-five years, who for twenty-one years had attacks of sharp pain in the right side of the lower part of the abdomen, the pain radiating through to the back and up to both shoulders and invariably associated with distension. About eight years ago he vomited during a severe attack, which lasted for five days; he suffered from constipation and frequent attacks of mild distension. He also complained of hot flushes of the face associated with frontal headaches.

Two days before operation he had an attack of pain in the abdomen and vomiting. On examination the abdomen was distended, visible peristalsis was present, and rigidity in the lower part of the abdomen. He had vomited brown fluid and his tongue was a dirty colour; his temperature was subnormal and the pulse rate was increased.

At operation on January 16, 1933, a Meckel's diverticulum was found. It was about 3·75 centimetres (one and a half inches) in length and was continued as a band crossing the ileum about 30 centimetres (twelve inches) from the ileo-caecal valve, and was then attached to the mesentery. A loop of bowel had become ensnared by this band, which was ligatured and divided. The diverticulum was then crushed near the base and removed, and the opening was closed with a double line of suture.

On January 17, 1933, the patient's abdomen was distended. A soap and water enema was given with a good result of formed fæces.

On January 18, 1933, the patient complained of pain in the abdomen, which was distended. A turpentine enema was given with pituitary extract; there was a good result with flecks of fæces.

On January 19, 1933, the enema was repeated with pituitary extract, and the rectal tube was used, both with fair result. Bismuth given on January 17, 1933, was recovered.

On January 20, 1933, the patient vomited a quantity of yellow fluid. A milk and treacle enema was given with a good dark brown fluid result.

On January 21, 1933, pain was present in the upper part of the abdomen, which was more distended; the patient looked ill and vomited a large amount of dark brown offensive fluid. Laparotomy was decided on and the abdomen was opened through a left paramedian incision. The bowel distal to where the Meckelian band crossed was collapsed, but no mechanical obstruction was

[1] Dr. Holmes wrote on August 2, 1933, that the patient's temperature began to rise eight days after the meeting. When it reached 38·3° C. (101° F.) he injected antistaphylococcal serum and the temperature became normal next day. A few days before Dr. Holmes wrote, the patient manifested polyuria; he passed approximately 5·4 litres (180 ounces) of urine; this amount was subsequently reduced to 3·3 litres (110 ounces).
Writing on August 8, 1933, Dr. Holmes said that the patient's temperature remained normal for five days. The patient then complained of pain in the right loin with painful respiration, and the rectal temperature rose again to 38·9° C. (102° F.). After another dose of serum the temperature fell, but did not become normal. The patient felt well and his general condition was much improved. Dr. Holmes thought that possibly the patient had a septic focus at the base of the right lung or in the diaphragm.

[2] The Acting Director of the Commonwealth Serum Laboratories informs us that the antistaphylococcal serum has antitoxic as well as antibacterial value.

present. An ileostomy was performed about five feet from this site. The fistula stopped discharging in about three weeks and the bowels began to act naturally with the help of enemata and later of their own accord. The wound healed in seven weeks.

(To be continued.)

Obituary.

WILLIAM ANGWIN EDWARDS.

DR. WILLIAM ANGWIN EDWARDS, who died, as previously recorded in these pages, on October 22, 1933, had achieved a reputation in the field of radiology. He was a recognized authority on radiography of the chest, and his death at the age of forty-one years has left a place that will be difficult to fill.

He went to school at Sydney Grammar School and studied medicine at the University of Sydney. He was admitted to the degrees of Bachelor of Medicine and Master of Surgery in 1915. After a short period spent as resident medical officer at Sydney Hospital he volunteered for active service. He went to England and obtained a commission in the Royal Army Medical Corps. In 1916 he went to France and carried out the duties of radiologist to Number 10 British General Hospital at Rouen. He held this position until 1918, when, owing to ill health, he returned to Australia. While he was in France he collaborated with Dr. Hull in the production of "Surgery in War"; he was responsible for the section of the book dealing with the localization of foreign bodies.

In July, 1918, he joined his brother, Dr. J. G. Edwards, in radiological practice at Sydney. He was appointed to the radiological staff of Sydney Hospital in 1919 and later became one of the senior radiologists. He also held appointments as radiologist at the Prince of Wales Hospital, Randwick, at Saint Vincent's Hospital, and at the Mater Misericordiæ Hospital.

Edwards did his most important work as a member of the Technical Commission of Inquiry into the incidence of chest diseases among miners at Broken Hill in 1921. He was also a member of the Commission of Inquiry into Diseases of the Chest among workers in Hawkesbury sandstone in 1926. It was the experience gained in these two inquiries that made him preeminent as a radiologist in chest conditions. His decisions were seldom called in question. At the time of his death he was attached to the Silicosis Commission, Sydney.

Edwards suffered for years from a distressing malady, but he was a cheerful person. Nothing seemed to depress him; his sense of humour was unusual, and it was always a delight to be in his company. He is survived by a wife and three children.

Dr. S. A. Smith writes:

W. A. Edwards was a brilliant radiologist and a man of outstanding charm. His colleagues on the Technical Commission at Broken Hill owed much to his skill and brilliance as a radiologist. Although a very young man on his appointment to this commission, his gifts were suited peculiarly to the work he was called upon to perform. Naturally quick-minded and endowed with good judgement, at once he understood and mastered the problems concerned in this radiological survey of the thousands of miners who were examined for the presence of chest diseases. His quick and objective mind led him straightway and surely to results which set a standard for this work in Australia, and this standard still stands, in spite of the mass of conflicting and changing opinions on occupational chest diseases. Thus it came about that, in addition to an extensive radiological practice, he was an acknowledged authority on these subjects. In his work on all occupational problems in their administrative and medico-legal aspects he retained always the most scrupulous fairness.

In addition to his eminence in his specialty, his personality was one of singular charm. In all circumstances, even in the face of constant ill health and the apprehension of serious catastrophes, he retained his gaiety and courage, and he leaves behind in the minds of his friends and colleagues the memory of a charming and brilliant man, whose life ended before he had reached his prime.

Correspondence.

DIATHERMY AND RETINAL DETACHMENT.

SIR: In your issue of August 19, 1933, page 258, column 3, E. F. King (*The British Journal of Ophthalmology*, May, 1933), the following appears in connexion with diathermy for retinal detachment: "The hole is localized by the simple expedient of marking at the limbus with India ink under direct ophthalmoscopy the meridian in which the hole is situated, and estimating in due (*sic*) diameters the distance of the hole in that meridian backwards."

But it is not so simple. Often it is very difficult indeed first to find the hole and then to map out its position. It has been a common experience at certain London hospitals to expend, a day or two before the operation, more than an hour, often two hours, and not uncommonly three, broken up where necessary into two or more sittings, before the hole had been found and it was felt that the line of the meridian had been so surely laid down and the distance along this meridian measured in disk-diameters to such exactitude that the surgeon could expect with a reasonable degree of confidence to find the hole beneath the point on the sclera thus indicated.

In connexion with the localization of the hole, no mention has been made of the following points:

1. That at the time of localizing the hole a counter ink mark must be made at the limbus opposite to the original

India ink dot, across which, at the time of operation, the guiding silk thread is carried to show the correct meridian. The method of carrying the thread across "the centre of the cornea", as estimated by the observer's eye, is unsatisfactory, as a very small deviation from the actual centre will give a considerable error in the supposed position of the hole if the latter is far back.

2. That in calculating the distance of the hole from the limbus it must be borne in mind: (*a*) That the *ora serrata* is one millimetre nearer to the limbus on the nasal side than on the temporal. (*b*) That the further back the hole is situated the greater must be the allowance for the curve of the eyeball, since the disk-diameter estimation is made on the retinal curve, whereas the measurement on the sclera is made by calipers which ignore the curve, this latter measurement being actually a straight line between the steel point at the limbus and the steel point on the sclera. If, therefore, the calipers were extended to the full disk-diameter estimation, the rear steel point would touch the sclera further back than the true position of the hole. This is quite a material difference when the hole is far back.

The use of Foster Moore's studs is not indicated in the diathermy technique, as the chorioid is not punctured until the diathermy needle has been laid aside.

Yours, etc.,

H. L. ST. VINCENT WELCH.

British Medical Association House,
135, Macquarie Street,
Sydney,
November 15, 1933.

AN APPEAL.

SIR: The Council of the Medical Benevolent Association of New South Wales has issued an appeal to the members of the medical profession in New South Wales for additional financial support, to enable it to give each beneficiary a present at Christmas time. The help given apart from this suffices merely for the bare necessaries of life; and at this season of the year it is felt that something more is called for.

The Council therefore appeals to all members of the medical profession in New South Wales for contributions to a fund for providing Christmas comforts and cheer to the unfortunate members of the profession and their dependants on its list.

All contributions should be sent to Dr. E. S. Littlejohn, Honorary Treasurer, 135, Macquarie Street, Sydney.

Yours, etc.,

ALFRED W. CAMPBELL,
Chairman.

135, Macquarie Street,
Sydney,
December 1, 1933.

CHRONIC NEPHRITIS.

SIR: Our attention has been called to a communication of Dr. Eustace Russell in THE MEDICAL JOURNAL OF AUSTRALIA of September 16, 1933, and to a letter of his in the journal of December 4.

We are surprised at the irresponsible way in which Dr. Russell throws doubt on the existence of lead poisoning amongst Queensland children. Were it not that he is senior physician to the Brisbane General Hospital, we would take no notice of either communication.

In the circumstances we are content to ask physicians to weigh the evidence (?) contained in both his communications and to weigh our published evidence, which has been accepted by many scientific physicians in Australia and in other countries.

Yours, etc.,

J. LOCKHART GIBSON.
A. JEFFERIS TURNER.

Brisbane,
December 5, 1933.

Post-Graduate Work.

POST-GRADUATE COURSE IN SYDNEY.

THE New South Wales Permanent Post-Graduate Committee announces that the general revision course for 1934 will be held in Sydney from Monday, May 21, until Friday, June 1, 1934. Those wishing to register their names for this course are requested to communicate with the Honorary Secretary, New South Wales Permanent Post-Graduate Committee, 225, Macquarie Street, Sydney.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on December 4, 1933.

Approval was given for the establishment of the degree of Master (M.Sc.Agr.) in the Faculty of Agriculture.

The following appointments were approved: Dr. H. V. D. Baret as Lecturer in Infectious Diseases in the Faculty of Medicine; Mr. A. W. Mackney as Demonstrator in the Department of Organic Chemistry; Miss C. Wedgwood as Lecturer in Anthropology for the first and second terms of 1934.

The several faculties reported to the Senate the election of Fellows and Deans for the two-year period commencing January 1, 1934:

Faculty of Arts: Professor F. A. Todd, B.A., Ph.D., as Fellow.
Faculty of Law: Professor the Honourable Sir John Peden as Fellow.
Faculty of Medicine: Dr. C. B. Blackburn, O.B.E., M.D., as Fellow.
Faculty of Agriculture: Professor R. D. Watt, M.A., B.Sc., as Fellow.
Faculty of Engineering: Professor W. A. Miller, M.E., B.Sc., as Dean.
Faculty of Science: Professor J. C. Earl, D.Sc., Ph.D., as Dean.
Faculty of Economics: Professor R. C. Mills, LL.M., D.Sc. (Econ.), as Dean.
Faculty of Veterinary Science: Professor J. Douglas Stewart, B.V.Sc., M.R.C.V.S., as Dean.
Faculty of Dentistry: Professor R. Fairfax Reading, M.R.C.S., L.R.C.P., L.D.S., as Dean.
Faculty of Architecture: Professor L. Wilkinson, F.R.I.B.A., F.I.A., as Dean.

Proceedings of the Australian Medical Boards.

VICTORIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1928, of Victoria, as legally qualified medical practitioners:

Cavanagh, Florence, M.B., B.S., 1933 (Univ. Melbourne), 311, St. Kilda Street, Brighton, S.5.
Gepp, Dorothy Marian, M.B., B.S., 1933 (Univ. Melbourne), "Strathalan", Greensborough Road, Macleod.
Lloyd-Green, Lorna, M.B., B.S., 1933 (Univ. Melbourne), "Dalkeith", 513, Mt. Alexander Road, Moonee Ponds.
Villiers, Alice Gwynne, M.B., B.S., 1933 (Univ. Melbourne), Hotel Warrnambool, Warrnambool.
Boan, Alan Ernest, M.B., B.S., 1933 (Univ. Melbourne), 24, Loch Street, St. Kilda, S.2.

Bowden, Keith Macrae, M.B., B.S., 1933 (Univ. Melbourne), 215, Plenty Road, Heidelberg, N.22.
Buckle, Donald Fergus, M.B., B.S., 1933 (Univ. Melbourne), Homœopathic Hospital, Melbourne, S.C.2.
Collins, Vernon Leslie, M.B., B.S., 1933 (Univ. Melbourne), Box 141, P.O., Nhill.
Cust, Norman Albert Austin, M.B., B.S., 1933 (Univ. Melbourne), 21, Bridge Street, Hampton, S.7.
Drury, Harrie Denison, M.B., B.S., 1933 (Univ. Melbourne), Ormond College, Carlton, N.3.
Duncan, Campbell Amiet, M.B., B.S., 1933 (Univ. Melbourne), 20, Lower Heidelberg Road, Ivanhoe, N.21.
Ebell, Frank Heinz, M.B., B.S., 1933 (Univ. Melbourne), Neerim Road, Oakleigh, S.E.12.
Fitzgerald, Leo Joseph, M.B., B.S., 1933 (Univ. Melbourne), 42, Abbotsford Street, Abbotsford, N.9.
Fulton, John Charles, M.B., B.S., 1933 (Univ. Melbourne), Cypress Street, Launceston, Tasmania.
Green, Ernest Joseph, M.B., B.S., 1933 (Univ. Melbourne), 27, Victoria Street, North Williamstown, W.16.

Books Received.

A SIXTH VENEREAL DISEASE, by H. S. Stannus, M.D., Ph.D., F.R.C.P., M.R.C.S., D.T.M. & H.; 1933. London: Baillière, Tindall and Cox. Demy 8vo., pp. 282, with illustrations. Price: 12s. 6d. net.

THE 1933 YEAR BOOK OF RADIOLOGY, edited by C. A. Waters, M.D., and I. I. Kaplan, B.Sc., M.D.; 1933. Chicago: The Year Book Publishers, Inc. Royal 8vo., pp. 804, with illustrations.

APPLIED PHARMACOLOGY, by A. J. Clark, M.D., F.R.C.P., F.R.S.; Fifth Edition; 1933. London: J. and A. Churchill. Demy 8vo., pp. 632, with 73 illustrations. Price: 18s. net.

THE QUEEN CHARLOTTE'S TEXT-BOOK OF OBSTETRICS; Third Edition; 1933. London: J. and A. Churchill. Royal 8vo., pp. 687, with four coloured plates and 301 text figures. Price: 18s. net.

A TEXT-BOOK OF BOTANY FOR MEDICAL, PHARMACEUTICAL AND OTHER STUDENTS, by J. Small, D.Sc., Ph.C., F.L.S., M.R.I.A., F.R.S.E.; Third Edition; 1933. London: J. and A. Churchill. Demy 8vo., pp. 727, with over 1,350 illustrations. Price: 21s. net.

LETTSOM: HIS LIFE, TIMES, FRIENDS AND DESCENDANTS, by J. J. Abraham; 1933. London: William Heinemann (Medical Books) Limited. Crown 4to., pp. 518, with illustrations. Price: 30s. net.

TEXT-BOOK OF PATHOLOGY, by R. Muir, M.A., M.D., Sc.D., LL.D., F.R.S.; Third Edition; 1933. London: Edward Arnold and Company. Royal 8vo., pp. 964, with illustrations. Price: 35s. net.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi and xvii

ANTI-TUBERCULOSIS ASSOCIATION OF NEW SOUTH WALES, SYDNEY, NEW SOUTH WALES: Honorary Physician.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer (male).
RENWICK HOSPITAL FOR INFANTS, SYDNEY, NEW SOUTH WALES: Medical Superintendent (male).
ROYAL ARMY MEDICAL CORPS: Medical Officers.
SAINT VINCENT'S HOSPITAL, MELBOURNE, VICTORIA: Medical Superintendent.
STATE PUBLIC SERVICE, BRISBANE, QUEENSLAND: Second Assistant Medical Superintendent.
THE ADELAIDE CHILDREN'S HOSPITAL (INCORPORATED), ADELAIDE, SOUTH AUSTRALIA: Resident Medical Officers.
THE AUSTRALIAN INLAND MISSION, SYDNEY, NEW SOUTH WALES: Medical Officer.
THE RACHEL FORSTER HOSPITAL FOR WOMEN AND CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Anæsthetists, Resident Medical Officer.
TOOWOOMBA HOSPITALS BOARD, TOOWOOMBA, QUEENSLAND: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chilliagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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